SHOUT
SHARE
IT

FOR THE PEOPLE
FOR EDVCATION
FOR SCIENCE

LIBRARY
OF
THE AMERICAN MUSEUM
OF
NATURAL HISTORY

LIBRARY
OF THE
AMERICAN MUSEUM
OF NATURAL HISTORY

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

59.06 (73

VOLUME XV

1917–1918

EDITED BY THE SECRETARY

NEW YORK

1918

LIBRARY
OF THE
AMERICAN MUSEUM
OF NATURAL HISTORY

18-78202. Dec. 16.

PRESS OF
THE NEW ERA PRINTING COMPANY
LANCASTER, PA.

CONTENTS.

PAGE

SCIENTIFIC PROCEEDINGS (85th–92d meetings):
- Communications of the eighty-fifth meeting 1
- Communications of the eighty-sixth meeting 15
- Communications of the eighty-seventh meeting 29
- Communications of the eighty-eighth meeting 52
- Communications of the eighty-ninth meeting 63
- Communications of the ninetieth meeting 81
- Communications of the ninety-first meeting 95
- Communications of the ninety-second meeting. 117

RECAPITULATION OF THE NAMES OF THE AUTHORS AND OF THE TITLES OF THE COMMUNICATIONS 147

EXECUTIVE PROCEEDINGS (85th–92d meetings) 157

REGISTER OF NAMES AND ADDRESSES OF THE MEMBERS 160

LIST OF OFFICERS 168

CLASSIFIED LIST OF MEMBERS 169

INDEX OF THE SCIENTIFIC PROCEEDINGS 172

SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

Eighty-fifth meeting.

Cornell University Medical College, October 17, 1917.

President Gies in the chair.

105 (1283)

The estimation of cholesterol in blood.

By **LUDWIG KAST, V. C. MYERS** and **EMMA L. WARDELL.**

[*From the Department of Medicine and Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.*]

The methods which are in use at present for the colorimetric estimation of cholesterol in blood are either time-consuming or of questionable accuracy. In connection with a study of the blood lipoids in obesity, it seemed necessary to investigate further the question of a suitable cholesterol method. For some time now we have employed a procedure which is comparatively simple and we believe to be very satisfactory.

The method consists essentially in mixing one c.c. of whole blood, plasma or serum with 4–5 grams of plaster of Paris, drying, and extracting the powder directly with chloroform in a special extraction apparatus. The plaster of Paris is emptied into a small paper extraction shell (4 cm. long) and this inserted in a short test tube (2.5 x 6 cm.) in the bottom and sides of which are a number of small holes. This is now attached to a large cork on a small reflux condenser and the tube and cork inserted in the neck of a 150 c.c. extraction flask containing about 20–25 c.c. of chloroform. Extraction is continued for 30 minutes on an electric hot plate, the chloroform made up to some suitable volume, such as 20 c.c., filtered if necessary, and colorimetric estimation carried out with the aid of the Liebermann-Burchard reaction, according

to the technique of Grigaut and of Autenrieth and Funk. Brown colors do not develop as with the Bloor method. An aqueous solution of naphthol green B, which is permanent, is used as standard.

Since the cholesterol esters give the color reaction, as pointed out by Bloor, saponification is unnecessary. With this method perfect duplicates and quantitative recoveries of added cholesterol and cholesterol esters may be had. The results are considerably lower than those obtained with the Bloor method.

106 (1284)

Morphin hyperglycemia as a test for pancreatic deficiency.

By JOHN AUER and ISRAEL S. KLEINER.

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute.*]

We found that the subcutaneous injection of one or two milligrams of morphin sulphate per kilo in dogs whose pancreatic substance had been strongly reduced by coagulation *in situ*[1] or by partial resection, caused a much greater rise in the blood-sugar level than the same dose in normal controls.

The following table gives the results of some of our experiments. It will be seen that the animals in which the pancreatic tissue had been reduced (AK5, 32, 37, and BD3) showed an increase in the blood-sugar three to four times greater than that obtained in the controls after the same dose of morphin.

As these animals with deficient pancreatic tissue may legitimately be considered in a prediabetic state, the morphin hyperglycemia observed in them may be of importance clinically in detecting individuals with an impaired carbohydrate metabolism. That this impairment need not be great and yet yield a strong hyperglycemia to a small dose of morphin is indicated by the fact that our dogs whose pancreatic tissue had been largely coagulated nevertheless showed a surprisingly good tolerance for sugar. In six tests where 10 grams of dextrose per kilo were fed, and in two where 4 to 5 grams of dextrose per kilo were injected subcutaneously, the amount excreted was nothing in two tests; less than

[1] J. Auer and I. S. Kleiner, these PROCEEDINGS, 1917, XIV, 151.

0.5 gram per kilo in three; less than one gram per kilo in two tests; and 1.5 grams per kilo in one test.

MORPHIN HYPERGLYCEMIA.

No.	AK5.	AK32.	Control 1.	Control 2.	AK5.	AK37.	Control 3.	Control 4.	BD3.	AK40.	Control 7.	Control 8.
Dose per kilo..	2 mg.	2 mg.	2 mg.	2 mg.	1 mg.	2 mg.	2 mg.	2 mg.	1 mg.	1 mg.	1 mg.	1 mg.
Normal.....	0.130	0.192	0.112	0.108	0.111	0.109	0.101	0.090	0.125	0.109	0.104	0.114
0.5 hr. after morphin....	0.206	0.230	0.097	0.111	——	——	——	——	——	——	——	——
1 hr. after.....	0.246	0.270	0.131	0.133	0.171	0.238	0.128	0.144	**0.213**	0.096	0.107	0.087
2 hrs. after....	**0.275**	**0.315**	0.128	0.151	**0.209**	**0.297**	0.124	0.138	0.197	0.141	0.136	0.115
3 hrs. after....	0.240	0.322	0.119	0.127	0.210	0.227	0.106	0.123	0.124	0.130	0.157	0.109
	All dogs fed 5 hours previously				Not fed to-day		Starved for 8 days		All dogs not fed to-day			

AK5: pancreas coagulated in situ with alcohol, 119 days before morphin test.

AK32: pancreas coagulated in situ with alcohol, 51 days before morphin test.

AK37: pancreas resected; uncinate process transplanted beneath skin 41 days before test.

BD3: five sixths of pancreas resected 90 days before; remaining sixth in connection with ducts.

AK40: uncinate process and tail of pancreas merely ligated 60 days before test.

Control dogs, normal animals, some were fasted.

Operated dogs all fed on regular *mixed* diet.

Blood samples from external jugular vein. Sugar determination by Myer's and Bailey's modification of the Lewis-Benedict method.

Whether the test will yield the same results in the human subject which we have obtained in dogs, can only be determined by trial, and such a trial we believe fully warranted by our findings. The procedure can easily be carried out with less than one cubic centimeter of blood if the Epstein method, for example, is used for determining the blood-sugar. The dose of morphin probably need not exceed 15–20 milligrams for an adult. The discomfort from the morphin will surely be considerably less than that following the ingestion of 1 to 200 grams of dextrose.

107 (1285)

Demonstration of the effects of castration on Seabright cockerels.

By **T. H. MORGAN.**

[*From Columbia University, New York City.*]

Two years ago I demonstrated before this Society castrated F_1 and F_2 cockerels that had been hen-feathered, but which in

consequence of the operation had, after moulting, begun to produce feathers characteristic of the normal male. Those birds have been kept to the present time, and now show a complete change to the cock-feathered type, except that the comb is reduced in size as in the ordinary capon. The spurs, however, appear to have their full growth. Since the F_1 birds were certainly heterozygous, and the few F_2 birds operated upon might have been heterozygous for cock feathering, it became desirable to carry out the operation on the pure Seabright cockerels which are always hen-feathered. The change in them, as the birds before you show, is as marked and as complete as in the F_1 and F_2 birds.

One bird after completely changing to cock-feathering (with reduced comb also) began after six months to change back; and, as is demonstrated by the bird, is at present in an intermediate stage. The comb has begun to enlarge again. Presumably a small piece of the testis was left—not large enough at first to prevent the change to cock-feathering—which has regenerated so far, that, at the last moult, the new feathers were affected.

108 (1286)

The effect of painting the pancreas with adrenalin on glycemia and glycosuria.

By I. S. KLEINER and S. J. MELTZER.

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

About fifteen years ago Herter and his coworkers reported that painting the pancreas with adrenalin causes a considerable glycosuria and they even assumed that the pronounced nature of glycosuria following intraperitoneal injections appears to be mainly attributable to the readiness with which the injected adrenalin comes in contact with the pancreas. Vosburgh and Richards reported later that the painting of the pancreas with adrenalin produces considerable hyperglycemia. To our knowledge the validity of these unusual statements was not confirmed or even tested since their publication. We have carried out recently a long series of experiments in which the hyperglycemia

and glycosuria were studied after painting the pancreas. In the main we could not confirm these claims, but we wish however to mention here only one striking result. In eight experiments in which the pancreas was so isolated from the peritoneum that none of the adrenalin could enter the peritoneal cavity, the hyperglycemia as well as the glycosuria produced by the painting was insignificant, surely not more than would have occurred if the adrenalin had been given subcutaneously or painted on some part of the peritoneum.

109 (1287)

Studies of dental caries, with special reference to internal secretions in their relation to the development and condition of dental enamel.

By WILLIAM J. GIES and Collaborators.

[*From the Biochemical Laboratory of the Schools of Medicine and Dentistry of Columbia University, College of Physicians and Surgeons.*]

A. Do substances in the blood pass from dental pulp into dental enamel of living animals? Hattie L. Heft.

B. Is dental enamel permeable to substances in saliva? Elizabeth C. Franke.

C. Effects of parathyroidectomy and castration on dentition in albino rats. Edgar G. Miller, Jr.

D. Dental effects of feeding glandular tissues to albino rats. Edgar G. Miller, Jr.

E. A new glycoprotein: dentomucoid. Leila Noland.

Decay of teeth, except that arising from trauma, may be due, primarily, to local deficiency in the structure and quality of enamel, or it may result from local specific disintegrative attack on enamel, regardless of normality of the enamel, or it may be caused by both these types of influences. In this series of studies we are endeavoring to ascertain whether "influences from the inside," such as those of a nutritional type and involving internal secretions, may be responsible factors in the incidence of dental caries.

Impairment of normal nutritive and endocrinic influences, *by subtraction*, has been induced by extirpation of various glands in albino rats. There were no effects on dentition after thymectomy, thyro-parathyroidectomy, and castration; deficient calcification of the teeth (incisors) followed parathyroidectomy.

Modification of normal nutritive and endocrinic influences, *by addition*, has been induced by feeding various glands to white rats. Dental calcification appeared to be (*a*) regularly *decreased* by oral administration of lymphatic, salivary, or thyroid, gland; (*b*) regularly *increased* by oral administration of testicle; and (*c*) wholly *unaffected* by oral administration of corpus luteum, parathyroid, pineal, pituitary, spleen, suprarenal, or thymus.

There were no effects on the general condition or dimensions of the teeth in any of the foregoing experiments.

Physiological variation in the composition of the teeth, in albino rats, is relatively slight and not great enough to account for any of the findings that were indecisive.

The well-known *chemical methods* we have employed were found to be adequate to detect significant chemical differences in dentition. Various results of uncertain import have not been due, at any time, to deficiencies in the analytic procedures as such.

Underlying all our experiments on the effects of internal secretions is the assumption that *chemical* changes may take place, *in developing enamel*, through the influence of substances that *originate* outside of, and enter, or superficially affect the cells involved in the production of enamel. If this assumption is unwarranted, it is obvious that internal secretions can have no *direct* chemical effect on the production or condition of enamel. If this assumption is incorrect, internal secretions can have, at most, only *indirect* effects on the development of enamel.

That our assumption in this general regard is correct, however, is shown by the fact that *trypan blue*, after its intraperitoneal injection into *young* rats, rabbits, and dogs, passes freely into the enamel of *developing* teeth, where the blue pigment seems to remain indefinitely; it does not pass from pulp through dentin into enamel of fully *erupted* teeth. (These particular facts were demonstrated at the meeting of the Society.)

That our assumption in this general regard is justified is shown,

also, by the fact that *strontium*, after its oral administration (as the chloride), daily for some time to *young* dogs, accumulates in the solid parts (and is present in the pulp) of the first and second sets of teeth, apparently taking the place of calcium. We have not yet determined whether strontium substitutes calcium, in such experiments, in the enamel of fully *erupted* teeth.

Experiments with *arsenic*, in the forms of salvarsan and arsenite (analogous to those with strontium), in an effort to determine whether arsenic can displace, or associate with, phosphorus in dental calcification, have given us wholly negative results.

Practically all the biochemical results referred to, thus far in this statement, were obtained on teeth *in process of development*. It has been cumulatively evident, as our experiments increased in number and scope, that internal secretions can have little or no effect, directly or indirectly, *on the enamel of fully developed and erupted teeth* (and therefore can have little or no continuing direct bearing on the problem of caries in such teeth) unless one or more *physiological* substances can pass from pulp through dentin into normal enamel, or from oral fluids into such enamel.[1]

We find that water passes freely back and forth through all parts, including the enamel, of fully developed natural extracted teeth. Simple mineral salts, such as sodium chloride, and common organic substances, such as cane sugar, show similar ability to diffuse back and forth through fully developed natural extracted teeth.

Our results in this general relation indicate that, whether or not there is true nutritive or maintenance *metabolism* in normal enamel, there may be physiological or pathological exchange of materials in enamel by *diffusion* from blood through dentin and enamel to oral fluids, and *vice versa*.

In the course of our study of the nature of tooth composition, from the point of view of effects of internal secretions on dentition (in their relation to the problems of cause, control, and prevention, of dental caries), we have found that teeth contain a *glyco-protein* that is closely similar to osseomucoid, and is evidently analogous to, though not the same as, salivary mucin.

[1] This statement ignores provisionally the possibility that caries may occur from changes in dentin that merely *undermine* enamel.

A complete study of the properties of this protein substance, which we have named *dento-mucoid*, is now in progress. We have already learned that it remains in teeth during the process of their acid decalcification. It is extractable, from decalcified teeth, with dilute alkalin solutions. It is precipitated, from such alkalin extracts, by mineral acids such as hydrochloric. It is an acid protein that forms colloidal salts. It yields reducing substance similar to glucosamin after acidic hydrolysis.

All of these studies are in progress, together with inquiries into the effects, on dentition in successive generations of albino rats, of treatment with thyroid, of unbalanced diets, and of toxic malnutrition.

110 (1288)

Diet and renal activity in tartrate nephritis.

By **WILLIAM SALANT** and **A. M. SWANSON.**

[*From the Pharmacological Laboratory, Bureau of Chemistry, Washington, D. C.*]

The subject of the experiments were rabbits that were fed oats or young carrots. Tartrate was administered to these animals either by mouth or subcutaneously, and the effect of the single dose as well as of repeated doses on renal activity studied by means of phenolsulphonephthalein. The following is a resumé of the results obtained.

1. When tartrate was given by mouth to rabbits on a diet of oats large doses were required to inhibit the elimination of phenolsulphonephthalein. The effects produced with medium doses were very moderate. Recovery was observed in all cases.

2. Even small doses of sodium tartrate injected subcutaneously into rabbits on a diet of oats caused a very pronounced inhibition of the elimination of dye. Considerable improvement occurred after 3 to 5 days, but complete recovery of function was never observed.

3. Evidence of disturbance of the renal function was seldom obtained with much larger doses of sodium tartrate when injected subcutaneously into rabbits on a diet of fresh young carrots. Large doses showed a decrease of functional activity within a few

hours after injection, but tests made one or more days later indicated considerable improvement, and in some cases, recovery.

4. After the subcutaneous injection of sodium tartrate into rabbits on a diet of oats the time of appearance of the phenol-sulphonephthalein injected was shorter and the duration of the elimination longer than in rabbits which had been receiving carrots.

5. When sodium tartrate was injected subcutaneously in gradually increasing amounts, no impairment of renal function was observed even with very large doses (4 and 6 grams per kilo) if the diet consisted of carrots exclusively, but the efficiency of the kidney was markedly decreased if oats alone were fed, although the amounts of tartrate administered was only one fourth or one sixth of that given to rabbits on a diet of carrots.

III (1289)

Experiments with an isomer of caffein.

By WILLIAM SALANT and HELENE CONNET.

[*From the Pharmacological Laboratory, Bureau of Chemistry, U. S. Department of Agriculture, Washington, D. C.*]

Physiological tests were made with 1 : 7 : 9-trimethyl-2 : 8-dioxy-purine. Its action was in every respect weaker than that of caffein. The toxicity was about one tenth that of caffein, the minimum fatal dose of 1 : 7 : 9-trimethyl-2 : 8-dioxy-purine for frogs being 4.0 to 4.5 grams per kilo. Renal activity as shown by experiments on rabbits, was only moderately stimulated in some cases and was very feeble in others although large doses were given intravenously. Perfusion experiments on the isolated frog heart indicated that an increase in force of cardiac action without altering the frequency may be caused by concentrations of 0.1 and 0.2 per cent. in Ringer's solution, but this effect was never very pronounced.

Prepared and kindly donated to this laboratory by Dr. C. O. Johns of this Bureau.

112 (1290)

The determination and significance of intragastric conductance.

By OLAF BERGEIM.

[*From the Laboratory of Physiological Chemistry, Jefferson Medical College, Philadelphia, Pa.*]

A retention stomach tube in the form of an electrolytic cell has been devised which makes possible the determination of intragastric conductances at any desired interval of time without disturbance or removal of gastric contents. The tip contains also a thermocouple which makes possible intragastric temperature determinations and corrections, and an aspiration tube by means of which samples of gastric contents may, if desired, be collected for analysis. By means of this apparatus intragastric conductance variations were studied in connection with determinations of total acidity, free hydrocholoric acid, pepsin, and trypsin.

The conductance of gastric juice is mainly due to the free hydrochloric acid which it contains and the same is generally true of the gastric contents. After the introduction of water or solutions (as sugar solutions) of very low conductance, the curve for conductance very closely follows the curve for free and total acid. This indicates that the equalization of osmotic concentration is brought about primarily by secretion of normal gastric juice.

After the ingestion of food containing protein the conductance curve usually lies below that for free hydrochloric acid as determined by titration because the latter values are high due to gradual dissociation of the protein salt. In the presence of weak organic acid as after fruit ingestion or of phosphate, as where much saliva is swallowed, the conductance falls below titration values and is a better measure of free hydrochloric acid.

Aside from the swallowing of saliva, the conductance of which is low, intragastric conductance is, after the first hour or so of digestion, almost always considerably modified by the regurgitation of pancreatic juice or bile or both and possibly to a lesser extent by pyloric and duodenal secretions. The conductance of pancreatic juice and bile being usually very low as compared with that of the gastric contents at maximum acidity, regurgitation

tends to markedly lower intragastric conductance as well as acidity. Conductance, however, rises relative to free hydrochloric acid on account of the higher salt content of these regurgitated secretions. After the ingestion of mineral acid, neutralization is brought about in the same manner as during digestion.

In achylias where intragastric digestion was mainly pancreatic in character, the conductance was found to parallel the concentration of pancreatic juice as measured by the tryptic index.

113 (1291)

The calcium content of the blood serum in certain pathological conditions.

By JOHN O. HALVERSON, HENRY K. MOHLER and OLAF BERGEIM.

[*Laboratory of Physiological Chemistry of Jefferson Medical College, Philadelphia.*]

The calcium content of human blood serum was determined in several normal cases and in a number of pathological conditions. In the normal cases values lying between 9 and 11 mg. of calcium per 100 c.c. were obtained. In nearly all of the pathological conditions studied, including cases where the blood clotted with extreme slowness, a similar range was observed, indicating a great constancy of this element in the blood serum. Distinct decreases were noted in cases of hematogenous jaundice, eclampsia, pneumonia, and particularly uremia. In several cases of uremia increases in serum calcium were noted on improvement in the clinical condition and following administration of calcium lactate. The urinary calcium excretion in severe nephritis was found to be low and calcium lactate administration brought about but slight absolute increases. Where marked general edema occurred, with or without nephritis, the excretion of calcium was unaffected by increased ingestion. In a case of pernicious vomiting of pregnancy with severe acidosis, alkali administration decreased calcium excretion to eight per cent. of its original value.

It is pointed out that as the red corpuscles are nearly free from calcium, determinations of this element in whole blood are of

little value, unless the relative volumes of plasma and corpuscles are known. It appears probable that the wide variations in the calcium content of whole blood in apparently normal individuals and variations for sex as reported by Lyman[1] may represent variations in corpuscular volume rather than in the calcium content of the plasma in these conditions.

ABSTRACTS OF THE COMMUNICATIONS, PACIFIC COAST BRANCH.

Eighteenth meeting.

San Francisco, California, October 3, 1917.

114 (1292)

Circulatory effects of tyramin.

By **A. W. HEWLETT.**

[*From the Division of Medicine, Leland Stanford Jr. University.*]

The subcutaneous injection of 0.06 to 0.08 gm. tyramin into normal individuals usually produces the following circulatory changes:

1. The systolic blood pressure increases markedly.
2. The diastolic blood pressure increases to a much less extent.
3. The pulse pressure is therefore increased.
4. The volume pulse in the arm becomes larger.
5. The heart rate is usually slowed.
6. The T wave in the electrocardiogram becomes notably larger with no constant change in the other waves. This alteration is most constant and is usually most marked in Lead II.

The changes in blood pressure and in the volume pulse in the arm indicate that the systolic output from the heart is increased. The changes in the electrocardiogram suggest those described by Rothberger and Winterberg after stimulating the right stellate (accelerator) ganglion.

Where epinephrin produces marked circulatory effects after subcutaneous injection these appear to be of a similar nature to

[1] Lyman, H., *Jour. Biol. Chem.*, 1917, XXX, 1.

those produced by tyramin. But tyramin is more constant in its action and it rarely causes the muscular tremor and apprehension that so frequently follows a large epinephrin injection.

115 (1293)

Absence of urea-splitting ferments in the animal tissues.

By AHMED E. SHEVKY (by invitation).

[*From the Laboratory of the Medical Division of Stanford University Medical School, San Francisco.*]

In 1912 Löb and Gutmann[1] published some data from which they concluded that a ferment capable of splitting NH_3 from urea existed in the pig's ovaries. Taniguchi[2] in a similar investigation which appeared last year confirmed this with somewhat more detailed data on determinations done with extracts from cow's ovaries. Both investigators used the Kruger and Reich method of ammonia estimation and their technique of extraction involved several hours of incubation.

In the present study corpora lutea were separated from fresh cow's ovaries, ground with twice its volume of 0.9 per cent. NaCl solution and filtered after standing one hour at room temperature. A similar extract was made from the rest of the ovaries. To 1 c.c. samples of a 2 per cent. urea solution were added 5, 10 and 15 c.c. of the filtrate and the mixture incubated for one hour at 37° C. Samples of urea solution and of the filtrate alone were similarly treated.

Ammonia estimations on all the samples were done by the aëration-titration method, using Barnett's recently described technique.[3] No more ammonia was found after incubation in the samples of extract and urea mixture than the combined ammonia content of the urea and of the extract samples.

In the following table figures for the corpora lutea extract are given, extracts from the rest of the ovaries gave similar results.

---

[1] Löb, W., and Gutmann, S., *Biochem. Z.*, 1912, XLI, 445.

[2] Taniguchi, Y., *Acta scholea med. univ. imp.*, Kioto, 1916, I, 3, 299.

[3] Barnett, G. D., *J. Bio. Chem.*, 1917, XXIX, 459.

2 per cent. Urea Sol., Cc.	C. L. Extract, Cc.	NH_3 in the Sample, Mgm.
1	—	0.051
—	5	0.076
1	5	0.119
—	10	0.136
1	10	0.170
—	15	0.143
1	15	0.196

Ammonium carbonate in passing through the liver is converted into urea. Urea on the other hand is converted into ammonium carbonate by an enzyme found in certain bacteria, fungi and a number of higher plants. In 1913 Fosse[1] published some data showing the occurrence of urea in fungi and higher plants, thus pointing to the possibility that the tissues of certain plants may be the site of both the formation of urea and its conversion into ammonium carbonate.

But all attempts to demonstrate the reversibility of the process whereby urea is formed in the animal body have so far failed. Earlier work on this subject reveals a number of conflicting observations. Perfusion experiments by Wakeman and Dakin[2] and by Jansen[3] on the liver under conditions which might be expected to favor a reversion of the reaction failed to show any conversion of urea into ammonia. Recently Barnett and Addis[4] found a marked increase of blood ammonia after intravenous injection of large doses of urea. Although this ammonia was shown to be at least in part due to bacterial decomposition in the intestine their experiments suggested the possible derivation of ammonia from urea in the body. But later experiments not yet published fairly establish the fact that the rapid rise of blood ammonia after the intravenous injection of large doses of urea is due solely to the bacterial action in the bowel.

The presence of urease in the ovaries—if proved—would have been strong evidence in favor of the theory of the reversibility of the ammonia-urea reaction in the body. The results of the experiments here reported fail to confirm the findings of the two earlier investigators.

---

[1] Fosse, R., *C. R. Academ. Sciences*, 1913, CLVI, 568.

[2] Wakeman, A. J., and Dakin, H. D., *J. Bio. Chem.*, 1911, IX, 327.

[3] Jansen, B. C. P., *Arch. Neerland*, 1915, II, 594.

[4] Barnett, G. D., and Addis, T., *J. Bio. Chem.*, 1917, XXX, 41.

SCIENTIFIC PROCEEDINGS.

Abstracts of Communications.

Eighty-sixth meeting.

New York Post-Graduate Medical School, November 21, 1917.

President Gies in the chair.

116 (1294)

A blood sugar tolerance test.

By **N. W. Janney** and **V. I. Isaacson.**

[*From the Montefiore Home and Hospital.*]

Various objections may be made to methods now in vogue for sugar tolerance determinations which depend on the appearance of sugar in the urine. Much more reliable, instructive and delicate results can be obtained by a study of the hyperglycemic response of the blood to ingested sugar. When pure glucose is administered in amounts equal to $1\frac{3}{4}$ gm. per kgm. body weight in 40 per cent. aqueous solution to fasting patients, alimental absorption is so regular that a normal hyperglycemic curve can be established. The blood sugar reattains its fasting level $1\frac{1}{2}$ to 2 hours after the sugar is taken. The oral method of administration is therefore much more certain than has been supposed.

For clinical purposes the test is carried out by first determining by Epstein's modification of the Lewis and Benedict method, the blood sugar of a patient who has fasted over night, then administering the sugar drink as prepared above, with the addition of the juice of a lemon and again determining the blood sugar at the end of two hours. In normal subjects the blood sugar has by this time returned to its fasting level. If hyperglycemia still persists, the blood sugar tolerance is lowered. This is the simplest form to which the technique is reducible. We prefer to make several half hourly or hourly observations of the blood sugar follow-

ing glucose ingestion also to determine the presence and amount of sugar voided in the urine during these determinations and in the complete twenty-four-hour specimen.

Glycosuria, which is normally absent under the conditions of the test, is not a necessary accompaniment of decreased tolerance. A normal blood sugar curve accompanied by glycosuria usually indicates renal diabetes. We have also found this test useful in the diagnosis of incipient diabetes. All cases of Graves' disease examined, and certain of chronic interstitial nephritis, show a prolonged blood sugar curve. Hypo-endocrine conditions such as cretinism and muscular dystrophy[1] are characterized by increased sugar tolerance with the ordinary urinary test, but blood sugar determinations showed hypoglycemia and a delayed tolerance curve. Evidently the hypoglycemia accounted for the contrary result obtained by the usual test. In view of these findings former clinical observations on sugar tolerance require revision.

117 (1295)

The inversion and determination of cane sugar.

By ANTON R. ROSE (by invitation).

[*From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.*]

Sucrose is completely inverted by heating in a solution of picric acid. This fact has been made use of in the estimation of cane sugar in solutions and extracts. After the cane sugar is hydrolyzed by heating with picric acid under properly controlled conditions, the invert sugar formed is determined by a modified Lewis-Benedict colorometric method. Glucose and fructose are determined at the same time.

The technic of the method is as follows: 1 c.c. of the clear liquid containing the sugars is transferred to each of two graduated narrow test tubes containing 2 c.c. saturated solution of picric acid. One of the tubes also contains 1 c.c. 20 per cent. sodium carbonate. The two tubes are then immersed into a bath of boiling water. After ten minutes 1 c.c. of 20 per cent. sodium

[1] Janney, N. W., Goodhart, S. P., and Isaacson, V. I., *Arch. f. Int. Med.*, article in course of publication.

carbonate is also added to the tube containing the acid mixture. After heating for 30 minutes the color in each tube has reached its maximum and they are allowed to cool. The contents of the tubes are diluted to a suitable volume and matched against a standard solution in a colorimeter. The readings for the tube which was alkaline at the beginning of the heating represents the glucose and fructose and the other tube represents these sugars plus the invert sugar from the sucrose.

When this method is applied to solids or semisolids, such as mashed fruit pulps, 1–10 grams are taken and triturated in a mortar with 100 c.c. water including the moisture of the sample and a clear liquid obtained by filtering or centrifuging.

Picric acid may also be used as the inverting agent in the determination of cane sugar by polarizing. The picric acid has no influence on the rotation of the polarized light and in many instances it acts as a clarifier and as a remover of soluble proteins. In practice it is well to add the picric acid in the form of a saturated solution, either one or two parts to each part of sugar solution to be determined, and to heat not more than twenty minutes in the boiling water bath. Glucose and fructose are both stable under these conditions.

118 (1296)

The influence of anesthesia and alkali therapy on the diastatic activity and sugar of human blood.

By JOHN A. KILLIAN (by invitation).

[*From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.*]

To study the effect of general anesthesia, produced by ether or chloroform, on the activity of the diastase and the sugar content of human blood, samples of blood were obtained from patients in the surgical service of the hospital, 12–24 hours before operation. A second sample of blood was drawn immediately after the operation, while the patient was still under the anesthetic. Determinations of the sugar content and diastatic activity, were made in these two specimens as described in an earlier com-

munication,[1] together with the CO_2 combining power according to the Van Slyke method. An increase in the diastatic activity with a parallel rise in the blood sugar was observed. The anesthesia was also found to produce a decrease in the alkaline reserve of the blood as shown by the CO_2 combining power.

A number of patients showing mild or severe hyperglycemia and increased diastatic activity of the blood received 20 to 30 grams of sodium bicarbonate by mouth per day. Changes in the diastatic activity, the blood sugar and CO_2 combining power of the blood were followed for some time. The results show that as the alkali reserve increased with the alkali therapy, the activity of the diastase declined, accompanied by a proportionate fall in the blood sugar content.

119 (1297)

Acid development as the result of injury in nervous tissue.

By **A. R. MOORE.**

[*From the Physiological Laboratory of Rutgers College, New Brunswick, N. J.*]

It was found possible to demonstrate the presence of acid in nervous tissue by the use of phenolsulphonephthalein as an indicator. The latter was employed in a 0.2 per cent. solution in Ringer's fluid, made slightly alkaline by the addition of one part of $M/15$ Na_2HPO_4 to four parts Ringer. Pieces of nervous tissue immersed in this solution take up sufficient dye in thirty minutes to show a striking reaction. In order to observe the reaction in brain substance, the brain of a frog was exposed by the removal of the roof of the skull, and the entire head immersed in the solution of phenolsulphonephthalein. When the preparation was examined thirty minutes later, the injured parts of the brain were bright yellow, indicating an acidity at least equivalent to PH = 6.5. The uninjured parts remained pink, but subsequent injury to them brought about the yellow coloration in a few seconds.

In similar fashion nerve trunks may be stained. The un-

[1] Killian and Myers: these Proceedings, 1916, XIV, 32; also Myers and Killian Jour. Biol. Chem , 1917, XXIX, 179.

injured parts are pink, but crushed areas and the cut ends are yellow. Microscopic examination showed the yellow color to be located in the fibers near the point of injury. If a nerve be crushed at a point previously uninjured, the development of acidity is shown at once by the change in color from pink to yellow. Stimulation of the nerve for five minutes with induction shocks causes the acid reaction to develop between the electrodes. Previous treatment with chloroform does not interfere with the reaction, but heating to 50° C. in physiological salt solution inhibits it. Killing the nerve with chloroform or by high temperatures does not cause the development of acid.

Assuming that the acid in question is either lactic or carbonic, it is possible to show that it is the latter. For this purpose, a method similar to that described by Haas[1] was used. Each tube contained 3 c.c. Ringer's solution, to which were added three drops of a 0.01 per cent. solution of the indicator. Carbon dioxide was completely driven out by aërating with carbon-dioxide-free air. The value of PH = 8.0. The tissue to be tested was put into the tube, the latter closed by clamping the short piece of rubber tube covering the end. The preparation was inverted for an instant, righted, placed in the colorimeter and a reading immediately taken. This reading is designated "start." Each subsequent reading was made in the same way. When a considerable amount of acid had been formed the tissue was removed and the solution aërated for thirty minutes with carbon-dioxide-free air. At the end of this time, in the case of nervous tissue, the H ion concentration had returned to the initial value, viz.: PH = 8.0. A piece of muscle run as a control gave off both lactic and carbonic acid, as shown by the fact that it was impossible to reduce the PH value to 8.0. These points are shown in the table, giving a set of measurements from the tissues of the same animal.

TABLE.

Brain, 72 Mg.	Sciatic Nerve, 87 Mg.	Sartorius Muscle, 63 Mg.
Blank.....PH = 8.0	Blank.....PH = 8.0	Blank.....PH = 8.0
Start.....PH = 7.8	Start.....PH = 7.8	Start.....PH = 7.8
10 min.....PH = 6.8	10 min.....PH = 7.35	10 min.....PH = 7.15
Aërated.....PH = 8.0	Aërated.....PH = 8.0	Aërated....PH = 7.6

[1] Haas, A. R., *Science*, N. S., Vol. 44, pp. 105–108, 1916.

120 (1298)

A new method of tissue culture for accurate and rapid measurements of the growth.

By **H. KRIGEL** (by invitation).

[*From the Department of Cancer Research of the Montefiore Hospital and Home.*]

The new method of culture consists in placing the tissue in capillary tubes instead of hanging drop, which was employed heretofore. The glass tube consists of two parts: one part, about 1 mm. in diameter, is used for handling by the operator, the other part, drawn out to about 0.3 mm. in diameter, is filled with the liquid medium, into which is placed the tissue to be cultivated. The thickness of the walls of this second part of the tube is less than 0.1 mm. The length of the whole tube is about 2 inches. 20 such tubes are placed with the thin ends down into a small beaker. A few drops of fluid are poured into the beaker, and this fluid through capillarity fills the tubes. The whole is placed into a test tube and may be sterilized and cooled without handling the individual tubes. A piece of tissue about 0.5 mm. in its widest diameter is placed into the wide part of each tube and then by the aid of a very fine glass rod immersed into the fluid, then with a piece of dry cotton attached to the thin end of the tube a drop of the fluid withdrawn and at the same time the tissue moved deeper in the fluid. The tissue becomes elongated and *grows only at both ends and consequently in one direction.* It is therefore quite easy to measure the longitudinal increase in size with accuracy of nearly $\frac{1}{20}$ of a millimeter. The narrow end of each tube is closed with sealing wax and the thick end by a gas flame. As a result of this manner of sealing the tube the piece of tissue falls out without effort on breaking off of the thin end of the tube.

For microscopical study 3 tubes are fastened to a microscopical slide and placed on a mechanical stage. The specimen may then be connected with a projection apparatus or a measuring device. The experiments were conducted with a solution con-

sisting of NaCl—1.2 per cent., CaCl—0.025 per cent., KCl—0.042 per cent., $NaHCO_3$—0.02 per cent. To 1,000 parts of this solution was added 1 part of commercial (3 per cent.) H_2O_2 in order to allow the tissue a sufficient amount of oxygen and at the same time obviate the appearance of free bubbles in the specimen. An increase of size of the tissue was obtained in nearly 85 per cent. of the specimens. Tissues used for the present study were the spinal cord and intestines of a chick embryo of 4 to 10 and 14 to 17 days respectively. The increase in size was pretty regularly about 0.5 mm. during the first twenty-four hours, which corresponds very nearly to the results obtained by M. R. Lewis.

The newly grown part of the tissue has often the same thickness as the rest of the piece, the whole piece becomes usually more uniform. On the other hand in about 36 specimens in which there was no growth the thickness of the tissue was not uniform. Apparently then a successful growth of tissue is not due to any chemical or physical phenomenon.

Experiments are undertaken with tissue cultures in serum and on influencing the growth with physico-chemical agents.

121 (1299)

Uridin and cytidin phosphoric acid.

By P. A. LEVENE.

[*From the Rockefeller Institute for Medical Research.*]

In a series of articles published in course of the last few years, Walter Jones and his co-workers advanced a theory on the mode of linkage of the four nucleotides taking part in the molecular structure of yeast nucleic acid. According to these authors, the nucleus of yeast nucleic acid is a tetra ribose of the following structure $[(C_5H_{10}O_5)_4—3H_2O]$. The assumption was based on the isolation of three substances which the authors viewed as dinucleotides, having the properties of a tetrabasic acid. In a previous publication the present author expressed the view that the experimental evidence adduced by Jones and co-workers was not sufficient to establish their theory. It also seemed to the present author that the experimental evidence presented was insufficient to establish the individuality of the guanosin

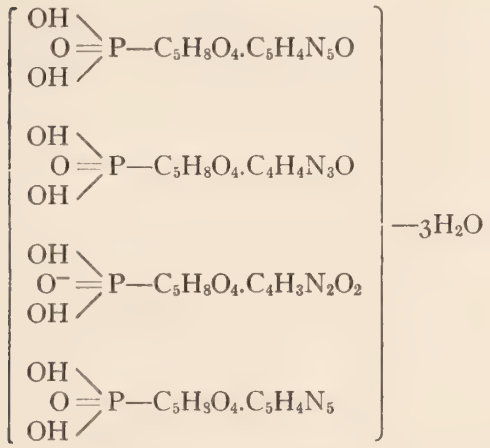
cytidine and adenosin-uracil dinucleotides. On the other hand, it seemed to the present author that a cytosin-uracil dinucleotide could actually be isolated from the mixture of pyrimidine nucleotides previously described by Levene and Jacobs.

The latter conclusion is now proven erroneous. Employing a different process for the fractionation of the brucine salts of the pyrimidine nucleotides it was possible to separate them into two brucine salts. One of these was converted into a crystalline barium salt of uridin-phosphoric acid, the other into an amorphous barium salt having the composition of the barium salt of cytidin-phosphoric acid.

The barium salt of uridin-phosphoric acid had a specific rotation of $[\alpha]^{20}_D = + 3.5°$. The barium salt as well as the products of hydrolysis with 10 per cent. sulphuric acid were tested for amino-nitrogen with a negative result. The base isolated on hydrolysis was pure uracil and the attempt to isolate cytosine-picrate from the mother liquor was unsuccessful.

The second brucine salt when converted into the barium salt gave a product which differed in composition and in physical properties from that of uridin-phosphoric acid. It deposited from a concentrated aqueous solution as a precipitate consisting of microscopic globules. It apparently had greater solubility and a higher optical rotation than the first salt $[\alpha]^{20}_D = + 14.0°$.

It is possible that this salt was not as yet obtained in the same degree of purity as the first. No dinucleotide was for the present isolated from the mixed brucine salts of the pyrimidine nucleotides. Of course, it is realized that eventually such conditions of hydrolysis may be found, that will yield dinucleotides. Efforts in this direction are now being made in this laboratory. However, for the present one must admit that the structural formulæ advanced by Jones and co-workers are merely a matter of speculation, and that the mode of linkage of the four nucleotides of the yeast nucleic acid still remains to be established. Omitting all arbitrary elements the structure of yeast nucleic acid should be represented as follows:



The barium salt of uridin-phosphoric acid had the following composition:

	Calculated. For $C_9H_{11}N_2O_9PBa$.	Found. I.	Found. II.
C	23.50	23.65	——
H	2.41	2.64	——
N	6.10	6.17	6.17
P	6.75	——	6.82
Ba	29.90	——	29.47
$Ba_2P_2O_7$	48.97	49.02	——

The specific rotation in 2.5 per cent. HCl was

$$[\alpha]^{20}{}_D \frac{0.14 \times 100}{1 \times 4} = + 3.5°.$$

On hydrolysis of the nucleotide the base uracil was obtained. It had the following composition:

Calculated. For $C_4H_4N_2O_2$.	Found.
N = 25.05	24.88

The barium salt of cytidin-phosphoric acid had the following composition:

	Calculated. For $C_9H_{12}N_3O_8PBa$.	Found.
C	23.56	23.71
H	2.64	2.55
N	9.16	9.39
P	6.77	6.43
Ba	29.96	29.36
$Ba_2P_2O_7$	48.97	48.79

The optical rotation of the substance was the following:

$$[\alpha]^{20}_{D} \frac{0.56 \times 100}{1 \times 4} = + 14.0°.$$

122 (1300)

The influence of X-rays on the development of the crown gall.

By **ISAAC LEVIN** and **M. LEVINE.**

[*From the Department of Cancer Research of the Montefiore Hospital and Home.*]

Crown gall is an infectious disease of plants which may be induced artificially by inoculating with the aid of a needle prick of a drop from an agar culture of *Bacterium tumefaciens*. Erwin F. Smith, of Washington, who investigated the condition continually for the last 10 years, is of the opinion that the disease is identical with human cancer. Irrespective of the stand one takes in regard to the identity of the two conditions, it must be conceded that there is a great deal of analogy between them. Crown gall as well as cancer is a new growth caused by a continuous, limitless proliferation of a group of cells within a tissue which normally do not proliferate at all. As a result of the rapid proliferation the new cells remain young and undifferentiated.

Clinical and experimental evidence indicates that the main biological and therapeutic action of X-rays consists in inhibition of the proliferating power of young undifferentiated cells. In man and vertebrates the mechanism of the action of X-rays on tumor cells is obscured by the changes in the cellular elements of the blood, lymph and the fixed connective tissue cells of the surrounding regions which is always encountered side by side with the changes in the tumor cells themselves. The crown gall, on the other hand, is an ideal subject for the study of the direct biological action of the X-rays on the tumor cells, since there is no other tissue present which may be changed by the rays.

For the present study ricinus plants and agar subcultures of *Bacterium tumefaciens* were used. The seeds for the former and the primary culture for the latter were obtained through the

courtesy of Dr. Erwin F. Smith. The plants were X-rayed by the modern methods, using hard rays. Preliminary experiments have shown that with the technique employed normal plants were not disturbed by the X-rays in their growth and development. Preliminary inoculation with the *Bacterium tumefaciens* have also shown that the inoculations were uniformly successful and were followed by the development of a large crown gall. 43 plants were inoculated with the microörganism and the inoculation was followed immediately by X-ray treatment. The treatment was given to each plant 6 times in the course of 2 weeks at intervals of 2 days. Simultaneously with the X-rayed plants, control plants of the same age and size were inoculated with the microörganism from the same subculture. All the control plants developed a large crown gall, the majority of the X-rayed plants on the other hand developed no growth, and only a slight swelling appeared at the place of inoculation. In 10 plants there developed a small stunted growth.

The microscopical study of X-rayed galls, a full report of which will be given later, indicates that the main immediate action of the X-rays on the cells of the crown gall consists not in a direct destruction of the cells, but in the arrest of the proliferating power. The analysis of the mechanism of the action of the X-rays on the crown gall seems to indicate at the same time that the rays influence and inhibit the functions of the cells directly and do not destroy the bacteria. But this phase of the subject will also be discussed in greater detail in a subsequent publication.

123 (1301)

The two forms of glycine.

By K. GEORGE FALK and KANEMATSU SUGIURA.

[*From The Harriman Research Laboratory, The Roosevelt Hospital, N. Y.*]

The two forms of glycine, plates from water, and needles from alcohol and water, were studied.

Emil Fischer[1] had shown that an acid chloride could be ob-

[1] E. Fischer, *Ber.*, 38, 2914 (1905).

tained from the latter, but not from the former. Further differences between the two forms were found on heating (decomposition temperatures), in the reaction with nitrous acid (van Slyke method for amino-nitrogen), and toward bromine.

124 (1302)

On the absorption of apomorphin and morphin through unusual channels.

By DAVID I. MACHT (by invitation).

[*From the Pharmacological Laboratory, Johns Hopkins University and the James Buchanan Brady Urological Institute, Baltimore.*]

The alkaloid apomorphin is well known as a typical centrally acting emetic, producing vomiting by the direct stimulation of the vomiting center. In case of dogs, as is also well known, morphin is found to produce emesis almost equally well. The author has taken advantage of these facts in the study of absorption of drugs through various unusual portals of entry.

On introducing a few milligrams of apomorphin in the form of powder or on instilling a few drops of a solution of 1 per cent. of that drug into the conjunctival sac, vomiting was found to be produced generally in from three to five minutes. Exactly the same phenomenon was observed after the administration of small quantities of morphin. These experiments indicate that the above drugs are easily absorbed through the eye into the general system. That this absorption is in part, at least, due to direct entry into blood and lymph channels and is not a result of an indirect absorption through the nasal ducts, was proven by obstructing the latter canal. This was done in two ways: in some cases the nasal duct was ligated and in the other cases it was occluded by cauterization. Even after obstruction of the nasal duct, apomorphin and morphin were found to produce emesis, though the process required a longer time.

In a similar manner apomorphin and morphin, when introduced into the vagina of dogs, can be shown to promptly induce vomiting, thus demonstrating their absorption through the vaginal wall.

Apomorphin introduced into the nasal canal of the dog, proper care being taken to prevent its reaching the pharynx, was also found to be absorbed and to produce vomiting.

In a similar manner the author has been able to show the absorption of apomorphin and morphin through the urethra, the prepuce and other structures. A comparative study of the absorptive powers of the urethra (in the male) and the bladder, is now in progress.

After having studied the absorption of apomorphin and morphin through the above-mentioned channels, the author has undertaken an extensive investigation concerning the absorption of a large variety of drugs and poisons through the same channels, the results of which investigation will appear in due time.

SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

Eighty-seventh meeting.

Rockefeller Institute for Medical Research, December 19, 1917.

President Gies in the chair.

125 (1303)

The solvent action of antiseptics on necrotic tissue.

By HERBERT D. TAYLOR and J. HAROLD AUSTIN.

[*From the Laboratories of the War Demonstration Hospital of the Rockefeller Institute, New York.*]

The work which we wish to report was undertaken with the idea of demonstrating the relative solvent action of the chlorinated antiseptics on necrotic tissue, pus cell, erythrocytes, plasma clot, and blood clot.

Liver tissue was purposely infected, placed in the incubator until thoroughly necrotic, shaken in salt solution with broken glass until emulsified, strained through one layer of gauze, and 5 c.c. portions added to bottles containing 50 c.c. of the solutions to be tested.

After shaking at half-hour intervals for two hours, 15 c.c. portions were removed and centrifuged for 5 minutes at the same high speed in each instance. The volume of sediment thrown down was measured, and by comparing the amount left after the action of antiseptic solution with that remaining after the action of control solutions of distilled water or salt solution, the amount of solvent action could be readily determined.

Dichloramine T is not soluble in water, and so oily solutions had to be used either superimposed on an aqueous suspension of the liver emulsion or alone.

These experiments showed that chloramine T and dichloramine T were without solvent action, while Dakin's hypochlorite solution in the concentration and degree of alkalinity used clinically readily dissolved the necrotic tissue. A similar action was demonstrated on pus cells, plasma clot, and red blood cells. Fiessinger (1) had already demonstrated this action on pus cells, but attributed it to the alkalinity of the hypochlorite solutions used. We have shown that this action was marked in a neutral solution of hypochlorite, whereas in a control solution without hypochlorite but of a degree of alkalinity comparable to Dakin's hypochlorite solution, it was wanting. Alkali added to the neutral hypochlorite enhanced its solvent action somewhat. Rous and Jones (2) have shown that intact leukocytes may protect virulent bacteria from the action of antiseptics. Hypochlorite solution by disrupting these cells will be able to reach and exert an antiseptic action on these organisms, while chloramine T and dichloramine T will have no such effect.

The solvent action of hypochlorite in the degree of alkalinity allowable for clinical use ceases below about 0.2 per cent. concentration of sodium hypochlorite. This point varies inversely with the alkalinity of the solution. None of these solutions had any solvent action on whole blood clot.

Curves shown by Carrel and Dehelly (3) demonstrate the ease with which infected wounds containing much necrotic tissue may be sterilized with the use of Dakin's hypochlorite solution.

REFERENCES.

1. FIESSINGER, N., MOIROUD, P., GUILLAUMIN, C. O., AND VIENNE, G. *Annales de Méd.*, 1916, III, 133.
2. ROUS, P., AND JONES, F. S. *Jour. Exp. Med.*, 1916, XXIII, 601.
3. CARREL, A., AND DEHELLY, G. The Treatment of Infected Wounds, New York, 1917.

126 (1304)

Differentiation of typhoid, paratyphoid A and B by means of a dextrin-inosite medium.

By FLORENCE HULTON-FRANKEL and KATHERINE MACDONALD (by invitation).

[*From Harriman Research Laboratory, Roosevelt Hospital.*]

Typhoid and paratyphoid *A* and *B* can be differentiated by means of a medium consisting of a 3 per cent. agar containing 1 per cent. of inosite and 1 per cent. dextrin, using litmus as an indicator. The dextrin must be one of the lower dextrins. The typhoid ferments the dextrin with acid formation in the butt of the tube, decolorizing entirely in 24 hours with a violet slant. Paratyphoid *A* does not ferment either dextrin or inosite and so the butt and slant of tube both remain violet in color. Paratyphoid *B* ferments inosite with gas formation and so paratyphoid *B* decolorizes the butt of the tube with the formation of gas bubbles, while the slant remains violet in color.

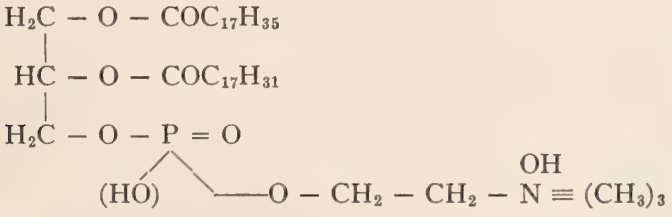
127 (1305)

Hydrolecithin and its bearing on the constitution of cephalin.

By P. A. LEVENE and C. J. WEST.

[*From the Rockefeller Institute for Medical Research.*]

The recent investigations on the chemical structure of lecithin have resulted in many important contributions, all of which point to the correctness of the generally accepted view of its molecular structure,



However, a scrutiny of all the work on lecithin reveals a remarkable incompleteness of each individual investigation. A

rigorous proof of the accepted theory requires an elementary composition of C = 65.70, H = 10.79, N = 1.74, P = 3.86. It further requires the nitrogen of the molecule to be composed entirely of choline. Hence lecithin should not contain even a part of its nitrogen in the form of free amino groups.

The work up to the present has satisfied many of the requirements. The fatty acids and the glycerophosphoric acid have been identified; and MacLean has prepared, at least once, a sample of lecithin that was free of amino-nitrogen. However, this one sample was very incompletely analyzed. All other samples of lecithin prepared by various workers contained amino-nitrogen in their molecule, and from the standpoint of elementary analysis showed a marked disagreement with the theory. This is well illustrated by the following table:

Author.	Source.	C.	H.	N.	P.	Choline, Per Cent.
Thudichum	Brain	66.75	18.67	1.81	4.00	.....
Baskoff	Liver	64.64	10.71	1.95	4.00	.....
Heffter	"	.....	.....	.....	.....	25
Stern and Thierfelder	Egg	64.63	10.96	1.79	3.95	.....
MacLean	"	64.18	10.60	1.87	3.95	66
Erlandsen	Heart	66.29	10.17	1.87	3.95	42
MacLean	"	66.27	10.32	1.85	3.97	41.4
Eppler	"	66.46	10.69	1.87	4.03	.....
MacLean	"	.....	.....	1.89	4.04	68
"	Not given	.....	.....	1.85	4.00	66
"	From $CdCl_2$ salt	.....	.....	1.87	4.15	98.7

The analytical data obtained by Ritter on hydrolecithin showed better agreement with the theory, and one might have been inclined to regard the material of Ritter as such that contained all the necessary and sufficient proof in favor of the conventional theory. Unfortunately, Ritter did not determine the amino-nitrogen of the reduced lecithin, and hence failed to furnish definite proof of its purity.

Indeed, the present report contains data unmistakably proving that hydrolecithin of an elementary composition fully harmonizing with the theory may be and generally is impure, containing between 10 and 20 per cent. of its nitrogen in the form of amino-nitrogen.

This finding has a great significance because of its bearing on the structure of cephalin, and the work is presented in its present

incomplete state because of this. On the basis of recent work on the hydrolytic products of cephalin a certain structural formula has been assumed. This formula requires an elementary composition of C = 66.17, H = 10.57, N = 1.88, and P = 4.17. However, all samples analyzed beginning with Thudichum and up to the present by most recent investigators consistently had the average composition of C = 60.00, H = 9.30, N = 1.80, and P = 3.80.

On the basis of these considerations, one may argue that if cephalin and lecithin both had the composition required for them by the theory, then a mixture of the two should possess practically the same elementary composition as either one of them in the pure state. On the other hand, if lecithin possessed the composition assumed by the theory and cephalin that found empirically, then a mixture containing 80 per cent. of the one and 20 per cent. of the other should possess a carbon content of about 64 per cent. Conversely, if a mixture of the two reduced substances possessed an elementary analysis of C = 65.3, H = 11.20, N = 1.75 and P = 3.85, as was actually found, it would justify the conclusion that both lecithin and cephalin possess the composition assumed for them by the theory.

The material analyzed by us contained 80 per cent. hydrolecithin and 20 per cent. of an impurity. It was found that the material yielded on hydrolysis besides the choline also the base aminoethanol which was isolated as the gold chloride salt. Hence it was reasonable to assume that the 20 per cent. of impurity consisted of hydrocephalin. If cephalin had the composition found by experience then a substance consisting of 80 per cent. of hydrolecithin and 20 per cent. of cephalin should have an elementary composition of C = 64.56, H = 10.49, N = 1.75 and P = 3.84. On the other hand if both lecithin and cephalin possess the structure assigned to them by theory then the above mixture of the reduced bodies should have the elementary composition found by experiment. Thus the facts presented in this report furnish evidence in favor of the prevailing theory of the molecular structure of lecithin and of cephalin. They also indicate the method by which the reduced cephalin may eventually be obtained. Efforts in this direction are now in progress.

128 (1306)

Fat emboli and shock.

By C. J. WIGGERS.[1]

[*From the Physiological Laboratory, Cornell University Medical College, New York.*]

Among the many explanations as to the cause of the circulatory failure in clinical as well as experimental shock, the theory that it is caused by fat embolism has been recently suggested. At least two distinct views as to the manner in which fat emboli may produce the circulatory failure are held: According to Porter (1), as we understand his view, fat, injected intravenously or gaining access to the venous circulation after fractures or laceration of the subcutaneous tissues, *passes through the pulmonary vessels but lodges in the peripheral systemic vessels and thereby produces circulatory failure by some mechanism as yet not clearly explained.* According to Bissell (2) the circulatory failure of postoperative or traumatic shock is caused by the lodgement of fat emboli in the pulmonary vessels, making it synonymous with pulmonary embolism.

During the past ten weeks we have re-investigated the following questions: (1) Is the mechanism by which the circulation fails after intravenous injection of oil the same as that following operation? (2) Is circulatory failure following fat injection primarily due to emboli of the pulmonary or systemic vessels? (3) Do fatty emboli of the systemic vessels produce circulatory failure similar to that following operation and trauma? To do this the mean pressure in the carotid artery, pulmonary artery and the effective pressures in the left and right auricles were studied in naturally breathing animals.

Experiments showed that when the circulation fails during shock produced by exposing the intestines, the pressures in the systemic and pulmonary arteries fall and a *marked reduction* of the actual, as well as the effective venous pressures in the right auricle takes place. These dynamic changes, which we regard

[1] This research was carried out in collaboration with Miss A. Kuehner, Messrs. H. Belcher, H. Cooper, W. Dodd, R. Douglass, M. Holsted and J. Sutton, Jr.

as characteristic of shock, do not occur when oil as such or in emulsion is injected intravenously. The only similarity consists in the fact that the mean arterial pressure falls. The pressure in the left auricle falls, but in the pulmonary artery and right auricle rises markedly. This can be interpreted to mean only that the failure of the systemic pressure is due to pulmonary emboli, a fact verified by microscopical examination of the lungs by Professor Ewing. As the venous and pulmonary arterial pressure changes are just the reverse of those found in shock, it is preferable, for academic as well as for therapeutic reasons, to distinguish circulatory failure produced by fat emboli from that due to surgical shock.

Although pulmonary embolism is apparently primarily responsible for the failure of arterial pressure when fat is injected intravenously, the fact that some of the oil passes through the pulmonary vessels and produces emboli in the systemic circuit (Bissell (2); Warthin (3); preparations kindly examined by Professor Ewing) raises the question whether such systemic emboli may not produce circulatory failure similar to that found in shock. To test this possibility, oils as such and in emulsions were directly introduced into the arterial circulation via the left brachial artery, thus avoiding pulmonary emboli. The effects of such injections were variable, depending apparently on where the fat emboli lodged. In some experiments large quantities of oil could be injected without any apparent effect on the arterial pressure In one case 24 c.c. of oil in emulsion and 8 c.c. as neutral oil were injected without any effect on the arterial pressure. In other cases the injection of oil produced a slight rise of arterial pressure, after which the pressure gradually recovered to normal. In a third group of cases the arterial pressure after a slight initial rise fell rapidly and the animal died within a few minutes. Electrocardiograms showed that in these cases the heart was not fibrillating but that the impulses were distributed in a normal manner to the last. Death was evidently due to respiratory failure brought about by fat emboli of the medulla. The pressure in the right auricle rose markedly, due to the cardiac failure following asphyxia. In none of these cases did the circulation fail as it does in shock.

In many instances the intra-arterial injection of oil is followed by a pronounced elevation of arterial pressure. When this occurs the effective venous pressure remains unchanged as long as the heart rate is not modified to a pronounced degree. In some cases the elevation of arterial pressure is accompanied by a marked slowing of the heart, giving the curve an appearance very similar to that following the injection of pituitary extract. From the high level thus reached the pressure gradually returns to normal and, after an hour or so, may reach a level considerably below normal. So far as our observations have gone, however, the pressure never falls as low as 50 mm. of mercury, while the venous pressure is either unchanged or elevated. The animal died in several hours of respiratory failure or from a progressive slowing of the heart, the significance of which has not been determined. The venous pressure at this stage is elevated.

Since neither the intravenous nor the intra-arterial injection of fat produces changes in the dynamics of the entire circulation which are comparable to those found in shock following exposure of the intestine, the hypothesis that fatty emboli, either of the pulmonary or systemic vessels, is the cause of circulatory failure in surgical or traumatic shock is not corroborated by our experiments.

REFERENCES.

1. W. T. PORTER. Fat Embolism a Cause of Shock, *Boston Medical and Surgical Journal*, 1917, *176*, 248; Observations sur le choc traumatique, *Compt. rend. Acad. d. sc.*, 1917, *165*, 164.
2. W. W. BISSELL. *Jour. Surg., Gynec. and Obst.*, 1917, *25*, 8.
3. A. S. WARTHIN. *International Clinics*, 1913, *4*, 171.

129 (1307)

Lantern slide demonstration of the effect of magnesium sulphate upon tetanus.

By **J. AUER** and **S. J. MELTZER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

Four pictures were shown. The first picture was that of a dog five days after it had received tetanus toxin subcutaneously

in the forehead. It showed extreme trismus, opisthotonos and (tonic) tetanus of all four extremities. The second picture was taken after the dog received intravenously a quantity of magnesium sulphate. The mouth was open, no opisthotonos, and the legs fairly relaxed. The third picture shows the same dog after it received a further quantity of magnesium sulphate. All tetanic signs were clearly abolished and the dog was lying on the back and looked well relaxed. In the fourth picture the dog was photographed on the floor. It was lying on the abdomen comfortably relaxed, the lower jaw slightly hanging down, but the head raised and in a normal position. Fifteen minutes later the dog was walking around in the laboratory without much stiffness.

The demonstration shows, first, that magnesium sulphate administered carefully intravenously in 6 per cent. solution is capable of abolishing all tetanic symptoms and, second, that the relaxing effect is not of a curare-like nature.

130 (1308)

Does the thymus gland of mammals when given as food to amphibians exert any specific influence upon the organism?

By **E. UHLENHUTH.**

[*From the Rockefeller Institute.*]

1. When thymus is fed to salamander larvæ, this gland does not exert any specific growth-promoting influence.

This is best shown in the curves obtained from the average sizes of four series of *A. tigrinum*. Two of the series were kept in high temperature and two in low temperature and in each temperature one series was fed on thymus and one on normal food (mainly earthworms). The thymus diet did not accelerate growth. (The same fact is shown with the aid of pictures.)

Why other writers have reported faster growth in thymus-fed amphibians will be demonstrated by curves obtained from the average sizes of four series of *A. opacum* (*A*, *B*, *C*, *D*, 1916). In all four series, the food was given in pieces of approximately the same size and exactly the same quantity. Since a piece of earthworm contains a great deal of indigestible matter, *i. e.*, soil, cuti-

cule, etc., the worm-fed animals actually received less food than the thymus-fed animals. Therefore, the thymus-fed series grew more rapidly than the worm-fed series. Had we not recorded the number of pieces which were fed to each series, we would have been led to the erroneous conclusion that thymus promotes growth in a specific way.

That thymus does not promote growth in a specific way is also seen from the two following curves obtained from two series (*A*, *B*, 1917) of *A. opacum*. Again in both series the food was given in pieces on a forceps; but to each animal food was given every day, until it would take no more food. Here the worm-fed animals grew even more rapidly than the thymus-fed series.

When the differences in the quantity of food become still greater, the animals show differences in size which are far greater than any reported due to thymus feeding. This is demonstrated by three curves obtained from the average sizes of three series of *A. opacum* (*C*, *D*, *E*, 1917).

These experiments show clearly that the quantity of food had a greater influence upon the growth of the salamander larvæ than the quality of food; amphibians in general react more promptly to small quantitative food differences than warm-blooded animals. Extensive experience with amphibians shows that out of a set of larvæ, certain individuals may take an exceedingly small amount of food, while in other sets, several animals take surprisingly large quantities of food from the very beginning. Differences in size like those produced artificially in the above series may, then, arise by spontaneous and uncontrolled action of the animals themselves and create the impression that a certain kind of food used in a certain experiment caused the difference.

2. Concerning development and metamorphosis, different species of even the same genus seem to react quite differently to the thymus.

In *A. tigrinum*, the thymus feeding produced neither acceleration nor retardation of metamorphosis, if we do not include, for the present, one animal to be mentioned later on. This is indicated in the curves *R*, *S*, *T*, *U*, 1917, and in six pictures.

In *A. opacum* the thymus diet caused a decided acceleration of the development of the gills and the skin (demonstrated by

pictures) and metamorphosis started earlier in the thymus-fed sets than in the worm-fed series. Yet as seen from the curves, the thymus-fed animals also grew more rapidly than the worm-fed animals. Still when they metamorphosed, they were smaller than the worm-fed animals at the time of metamorphosis. It is only recently that I have felt the necessity of knowing more about the relation between metamorphosis and growth in amphibians, and only when our experiments on this point are completed will it be known definitely whether or not the thymus feeding actually does accelerate metamorphosis in salamanders.

The experiments just mentioned are also confusing on account of the fact that only part of the thymus-fed animals actually metamorphosed earlier than the worm-fed animals. In each series of the *Opacum* group there were several individuals which, after they had arrived at the stage preceding metamorphosis, suddenly stopped their development and either died or metamorphosed only after a certain period had elapsed.

In the *A. tigrinum* set there is one thymus-fed animal, which did not metamorphose when the others did. It is still a larva—10 weeks after the rest of the series metamorphosed.

In a thymus-fed series of *A. punctatum*, two individuals remained as larvæ for almost 15 months; and died upon commencing to metamorphose.

It is important to note that "long time" larvæ occurred mostly in thymus-fed series which were kept in low temperatures.

One cannot, of course, but recognize the important bearing on the problem in question, of these "long-time" larvæ, and an examination of them will be necessary. Yet it does not seem that the phenomenon has anything to do with the thymus; for entirely similar cases are encountered in normally kept animals. We had, for instance, one worm-fed animal among a series of *A. punctatum* kept in a cool temperature, which was still a larva 8 months after hatching and then unfortunately was killed by an unsuccessful operation. In another series of only 4 *A. punctatum* larvæ, kept in high temperature, there was one individual which grew normally but did not show any signs of development on its skin throughout its life; it remained larval when the other animals metamorphosed and died as a larva one month later.

3. Finally, attention may be called to an action of the thymus gland which was as yet unknown. Exclusive thymus diet produces in *A. opacum* and *A. punctatum* severe attacks of convulsions, similar to those reported in mammals after extirpation of the parathyroids and known as tetany. Larvæ of *A. opacum* are demonstrated in the state of an acute attack.

No definite idea about the relation between the thymus gland and these convulsions could be formed as yet; perhaps the most interesting characteristic of the phenomenon is that it always seems to start when the animals reach a certain stage of development, and the acute attacks cease when the animals are ready for metamorphosis. Metamorphosed animals of about 18 months which have been fed from the fourteenth day after birth exclusively on thymus, are shown; the animals do not exhibit any signs of convulsions.

From this it is certain that the thymus contains a substance which produces convulsions; whether or not it becomes effective seems to depend on certain organs which disappear or develop in the course of the development of the organism. In fact, there are some indications that the parathyroids are involved in this process: for, first, convulsions are not produced by thymus feeding in the frog and toad larvæ, which develop the parathyroids in a very early stage; and second, the acute convulsions in the *A. opacum* and *punctatum* larvæ cease approximately at the time when the parathyroids develop.

131 (1309)

Demonstration of blood from an extreme case of lipemia in diabetes mellitus.

By **A. I. RINGER.**

[*From the Chemical Laboratory of the Montefiore Home and Hospital.*]

The blood of a very severe case of diabetes was demonstrated; it contained 2.14 per cent. cholesterol and a total fat content of 14.4 per cent.

132 (1310)

A note on the preparation of Dakin's hypochlorite solution.

By GLENN E. CULLEN and JAMES H. AUSTIN.

[*From the War Demonstration Hospital of the Rockefeller Institute.*]

In the preparation of Dakin's hypochlorite solution it has been customary to adjust the reaction by the use of solid phenolphthalein. In the preparation from sodium carbonate and bleaching powder Dakin[1] added boric acid to the strongly alkaline hypochlorite solution until it no longer colored solid phenolphthalein. Daufresne[2] modified this technique by using a mixture of sodium carbonate and sodium bicarbonate for decomposing the bleach and also used solid phenolphthalein as the test for the reaction of the solution.

Investigations in this laboratory indicate that the reaction at which solid phenolphthalein turns red with a hypochlorite solution is at a hydrogen ion concentration of about 1×10^{-10} (pH = 10), *i. e.*, an alkalinity in terms of hydroxyl ions about 1,000 times that of water. Alkaline phenolphthalein solutions show a flash color in hypochlorite solutions at a hydrogen ion concentration of about 1×10^{-8} (pH = 8) or in the same unit an alkalinity 10 times that of water or one one-hundredth that of the turning point of solid phenolphthalein in hypochlorite solution. Hypochlorite solutions at a reaction corresponding to the color change of phenolphthalein solution, *i. e.*, pH = 8, are not stable. It is desirable therefore to find the minimum alkalinity at which a hypochlorite solution can maintain its concentration for a convenient period. We have found that this point is at a hydrogen ion concentration of about $10^{-8.5}$ (pH = 8.5). At this reaction a solution will maintain its hypochlorite content within the desired range for about two days. Hypochlorite solutions prepared by the use of phenolphthalein may vary as much as one hundred times in alkalinity, depending upon the bleach and conditions. This explains the variations in irritation that different observers have reported.

[1] Dakin, H. D., *British Med. Journal*, August 28, 1915.

[2] Daufresne, M., *Presse med.*, 24, 474, 1916.

We have developed the technique and tables for preparing hypochlorite solutions at the desired reaction from any bleaching powder, containing more than 20 per cent. available chlorine, in accordance with the following outline. The bleaching powder is decomposed with just enough sodium carbonate to insure complete precipitation of all the calcium, the filtrate is neutralized to solid phenolphthalein with dilute (10 per cent.) hydrochloric acid and the solution is then brought to the desired reaction, and a sufficient amount of buffer salts provided, by the addition of a definite volume of sodium bicarbonate solution.

We have also determined the amounts of carbonate and the technique for preparing hypochlorite solution of the desired reaction from liquid chlorine. We have been assisted in this by two engineering firms who have adopted their chlorine measuring devices to this purpose.[1] The method consists of running chlorine gas into a solution containing a weighed amount of carbonate until the desired percentage of hypochlorite is reached. The solution is then of correct reaction and contains a sufficient amount of buffer. We believe that on the basis of convenience, economy and accuracy that this is the method of choice.

We have moreover found that the indicator o-cresol-phthalein[2] changes color in hypochlorite solutions at about the "ideal" reaction and believe we can still further simplify the preparation by its use.

The methods we have indicated have been in use at our hospital for over two months and have proven themselves to be entirely satisfactory. They have also been found to be of such simplicity that they may be easily mastered.

---

[1] Wallace and Tiernan Co., New York; The Electro Bleaching Gas Co., New York

[2] Clark, W. M., and Subs, H. A., *J. Inf. Diseases*, 2, 1, 1917.

133 (1311)

Therapeutic experiments with Rosenow's antipoliomyelitic serum.

By HAROLD L. AMOSS and FREDERICK EBERSON.

[*From the Laboratories of the Rockefeller Institute for Medical Research, New York.*]

Opinions of bacteriologists on the etiology of poliomyelitis divide them into two well-defined camps. One group affirms that the streptococci bear a causal relationship to poliomyelitis and are biologically akin to the globoid bodies of Flexner and Noguchi; the other group denies that they are of essential etiologic importance and regards them as secondary invaders. The question is important because of its relation to the problems of prevention and treatment of the disease.

Because of the failure to produce in large animals a serum possessing therapeutic properties for the treatment of poliomyelitis, serum derived from recovered cases has been used. A far greater measure of success has recently been claimed in treatment of human cases by the use of serum produced in animals such as the horse by the repeated intravenous injections of streptococci. Obviously the treatment of a long series of cases from several epidemics is necessary before any definite conclusion can be reached concerning the efficiency of a specific serum. In the present case, however, we believe that the question may be more expeditiously answered by the experimental method.

An injection of minute amounts of active poliomyelitic virus intracerebrally into the monkey invariably results in paralysis and generally in death of the animal, but intravenous injections of much larger amounts of the same virus produce no symptoms. If, however, at the time of the intravenous injection or a few hours before, the meninges and choroid plexus are inflamed by the introduction of small amounts of sterile monkey, horse or human serum, or even sterile isotonic solutions of electrolytes, the virus passes from the blood into the nervous tissues and induces characteristic changes which lead to paralysis and death. Flexner and Amoss have shown that repeated injections of immune

serum invariably offset the effects of aseptic inflammation, probably by neutralizing the virus as it passes through. Two separate sets of experiments were carried out with Rosenow's serum, normal horse serum, and immune monkey serum, in order to determine whether the former contained neutralizing substances rendering it suitable for therapeutic application. The results are clearly decisive.

Experiment I, Nov. 12: *Macacus rhesus A* (control) received 50 c.c. of clear supernatant fluid obtained by centrifuging a 5 per cent. suspension of fresh active poliomyelitic tissue. The monkey remained well.

Macacus rhesus B. 5 p.m., November 11, received intraspinally 3.0 c.c. normal horse serum. November 12, 11:15 a.m., received 50 c.c. of the same virus suspension as Monkey *A* received. At 12 m. 3.0 c.c. normal horse serum were injected intraspinally. Repeated daily intraspinal injections of 3 c.c. normal horse serum were made until November 17, when the animal became slow. Later in the same day the animal became prostrate and died November 18. Typical lesions of poliomyelitis.

Macacus rhesus C received at 5 p.m., November 11, 3.0 c.c. activated Rosenow's horse serum intraspinally. On November 12 the intravenous injection of 50 c.c. of virus suspension was given, followed by intraspinal injection of 3.0 c.c. activated Rosenow's serum. The intraspinal injections were repeated daily. This monkey became slow on November 17, excited on November 19. On November 20 it dragged the right leg and climbed awkwardly. November 23, legs were paralyzed and deltoids weak. November 24, prostrate. November 26 moribund; etherized. Typical lesions of poliomyelitis.

Macacus rhesus D received at 5 p.m., November 11, 3.0 c.c. of pooled serum from monkey which had been paralyzed, recovered and reinforced by subcutaneous injections of active virus suspensions. On November 12 Monkey *D* received an intravenous injection of 50 c.c. of virus suspension, followed immediately by an intraspinal injection of immune serum. The intraspinal injections of the immune serum were continued for 6 days. The monkey remained well.

Experiment II differed from Experiment I only in the amount

of serum injected intraspinally. Instead of 3.0 c.c. of normal horse serum, Rosenow's serum, or immune monkey serum, 2.5 c.c. were used. The results were identical with those obtained in Experiment I.

The results of these two experiments unequivocally demonstrate that Rosenow's serum is devoid of protective power against poliomyelitic virus, while serum from paralyzed monkeys possesses perfect protective power, as has been shown previously by Flexner and Amoss.

There is a further corollary to this general deduction. Once it is established that the antibodies yielded by streptococci differ essentially from those induced by poliomyelitic virus, the contention that poliomyelitic virus and streptococci are identical becomes untenable.

134 (1312)

On the comparative absorptive power for drugs of the bladder and urethra (male).

By DAVID I. MACHT.

[*From the Pharmacological Laboratory and the Brady Urological Institute, Johns Hopkins University.*]

In other communications dealing with the absorption of drugs from the conjunctiva[1] and from the vagina,[2] published elsewhere, the author called attention to the fact that apomorphin, by virtue of its being a centrally acting emetic, furnishes a convenient means of demonstrating absorption of drugs through unusual channels. If a 1 per cent. solution of apomorphin hydrochloride is introduced into the bladder of a male dog through a hard catheter, the latter instrument being allowed to remain in place, the solution remains in the bladder and owing to the powerful spasmodic contraction of the urethral sphincter in the male dog, practically none of the drug gets into the urethra. Under these circumstances vomiting may occur not sooner than half an hour after the introduction of the poison and sometimes after the lapse of an hour

[1] *Jour. A. M. A.*, 1917, LXVIII, p. 1230.

[2] *Jour. of Pharmacol. and Exp. Therap.*, Vol. X, 1918, p. 509.

or more, and very often not at all unless the catheter be removed. If, on the other hand, the urethra of the same dog, on another day, be irrigated with the same solution or even weaker solutions of apomorphin, care being taken not to inject the drug into the bladder but to confine the irrigation only to the urethra and allow the fluid to run back, vomiting is produced in every case in from three to five minutes. Inasmuch as vomiting is produced in dogs almost as efficiently by means of morphin as with apomorphin, the same results can be obtained by using that alkaloid. Even strong solutions of morphin confined to the bladder produce either no vomiting at all or only after the lapse of a considerable period of time (half an hour to one hour). On the other hand, the introduction of a little morphin solution into the urethra is followed in the dog by vomiting in a few minutes. The remarkable difference in the absorptive power between the urethra and the bladder noted after morphin and apomorphin, holds good for a large number of other drugs and poisons. The author has studied in this connection the effect of various alkaloids, a number of antiseptics, some local anesthetics and a number of salts. The complete account of the investigation will be published in due time in the *Journal of Urology*. It may be stated in this place that an inquiry into the absorptive power of the ureters is also under investigation by the author.

135 (1313)

On the influence of some opiates and antipyretics on the field of vision.

By DAVID I. MACHT, S. ISAACS, and J. P. GREENBERG.

[*From the Pharmacological and Psychological Laboratories, Johns Hopkins University.*]

While the effect of drugs on the acuity and field of vision has as yet not been the subject of extensive study, the work extant, such as, for instance, that of Dreser[1] on the influence of strychnin on the visual function, indicates that important changes in visional perception may be produced by the ingestion of pharmacological agents. In connection with an extensive study of the effect of various antipyretics on different psychological functions, the

[1] Dreser, 1894, XXXIII, 251.

authors have made perimetric observations for the purpose of determining whether the various drugs used exerted any influence on the field of vision. The antipyretics studied and taken by month were: Acetanilid in doses of 5 to 8 grs., acetphenetidin in doses of 5 grs., antipyrine, in doses of 4 to 8 grs., quinine in doses of 5 grs., pyramidon in doses of 5 and 6 grs., aspirin in doses of 5 and 10 grs., salol in doses of 5 and 8 grs., and certain combinations such as acetanilid plus salol, 5 grs. each; aspirin plus salol, 5 grs. each; aspirin and antipyrine, 5 grs. each; acetphenetidin plus salol, 5 grs. each, and acetanilid and acetphenetidin, 5 grs. each. The effect of these antipyretics was then compared with the action of morphine and a combination of total opium alkaloids (pantopon) administered hypodermically in ordinary therapeutic doses to the same subjects on different days. The experiments were made on the authors themselves and a few of their colleagues with an ordinary perimeter, testing the field of vision for four colors, namely, white, blue, red and green.

It was found that the opiates, morphine and pantopon, taken by injection in every case produced a definite though very slight contraction of the field of vision. As between morphine and pantopon, little difference was noted; yet it may be well to state that in two out of three subjects on whom the observations were made, the morphine injections seemed to produce a slightly greater limitation of the field of vision than pantopon.

Of the antipyretics studied it was found that they produced either no change in the field of vision at all or had a slight tendency to increase it. This was especially noted in case of acetanilid, acetphenetidin, aspirin, and the combinations acetanilid plus salol and acetphenitidin plus salol, which in some of the experiments produced a definite though not marked increase in the field of vision. It was curious to note, furthermore, that the increase was especially apt to occur in case of the white and blue colors, which ordinarily under normal conditions give the largest field of vision.

The conclusions drawn from some of the observations are that the opiates, while producing but little effect upon the field of vision in the normal subject, when they do exert any influence, tend to narrow the field; the antipyretics, on the other hand, when any effect is to be noted, tend to increase the field of vision.

What mechanism may be producing the positive findings in the different cases, it is difficult to say. At any rate, various factors, such as the constriction of the pupil in the case of opium, vasomotor changes produced by antipyretics, specific effect upon the retinal ganglia and nerves and central cerebral effects must be all considered.

136 (1314)

The relation of growth and swelling of plants and biocolloids to temperature.

[*From the Desert Laboratory, Tucson, Arizona.*]

By **D. T. MACDOUGAL.**

The varying constitution and resultant water-relations of the plants may be simulated by mixtures consisting of agar and albumen, or albuminous derivatives, or most successfully by proteins extracted from beans or oats.

The swelling of living and dried sections of plants and of dried plates of such biocolloids in various solutions has been found by the author to depend upon the history or previous treatment of the material, the proportions of protein or albuminous derivatives, and pentosans present, concentration of the solutions and the temperature.

According to Taylor heat should diminish imbibition in gels, while cold and pressure will increase it.[1] The velocity and swelling capacity of two biocolloids at temperatures within the range of growth of plants used for comparison are illustrated by the following auxographic measurements expressed in percentages of increase of the original diameter of sections of dried plates:

SWELLING OF THIN PLATES OF AGAR, 90 PARTS, BEAN-PROTEIN 10 PARTS AND NUTRIENT SALTS .8 PART.

Increase in thickness in percentages of the original.

Temp.	Duration of Swelling.			
	4 Hours.	8 Hours.	10–12 Hours.	20–22 Hours.
15–16° C.	888%	1,113%	.....	1,325%
21–23° C.	1,388	1,550	.....	1,900
30–31° C.	1,472	1,735	.....	2,022
39° C.	2,486.1	2,736.0	(12 hours) 2,791	Complete
46–47° C.	2,153	2,347	(10 hours) 2,361	Complete
48–49° C.	2,361	2,514	Complete	.....

[1] Taylor, "The Chemistry of Colloids," p. 155, 1916.

The sections swelled at temperatures of 15–23° C., were .20 mm., those at higher temperatures, .18 mm. in thickness.

Plates of agar, 90 parts and oat-protein, 10 parts, show the highest hydration capacity of any mixture tested, the measurements obtained being as follows:

Temp.	4 Hours.	8 Hours.	10–12 Hours.	20–22 Hours.
15–16° C., .18 mm.	1,167%	1,459%	.....	1,794%
22–23° C., .18 mm.	1,388	1,550	.....	1,900
30–31° C., .16 mm.	2,172	2,763	.....	3,078
38–39° C., .18 mm.	2,541.6	2,861	(12 hours) 2,791%	Complete
46–47° C., .18 mm.	2,555.5	2,833.1	(10 hours) 2,361%	Complete
48–49° C., .16 mm.	1,906.3	2,031	Complete	.....

The swelling of discs 10–12 mm. in thickness, cut from joints of *Opuntia*, in water and salt solutions in illustrated by the following measurements:

Percentage of increase and duration of swelling.

	Dist. Water.	Potass. Nitrate, .01 *M.*	Potass. Nitrate Citric Acid, .01 *M.*
18–20° C.	11.8% (42 hrs.)	14% (42 hrs.)	8.8% (32 hrs.)
24–25° C.	13.6% (40 hrs.)	14.4% (42 hrs.)	13.2% (42 hrs.)
44–45° C.	7.6% (5 hrs.)	8.4% (7 hrs.)	4.9% (2.5 hrs.)

The swelling of the biocolloids is seen to increase in initial velocity and total amount to a maximum between 39° and 46° C. in the salted mixture, and to 46° C. in the unsalted plates. The sections of plants increase similarly to a point undetermined and the swelling may be much greater in preparations freed from mechanical tissues. These increases are parallel to accelerations and retardations of growth by living plants under the influence of temperature.

The hydration in question constitutes 97 per cent. or more of the volume increases known as growth and manifestly may not be regarded as the form of imbibition to which Chatelier's theorem may be applied simply as has been attempted by Taylor and others. The relation to swelling demonstrated in the above tables is one which finds a parallel in the action of agar-agar in my experiments. The high swelling coefficients displayed by the mixtures are probably associated with or due to the fact that the

two main colloids are unequally distributed in the two phases of the system. True *adsorption* must as Zsigmondy has pointed out[1] play an important part but the reactions imply that *absorption* also takes place. The arrangement of the denser continuous phase of the gel in the form of a fine sponge or of a close mesh of plates or fibers would offer conditions in which both adsorption and absorption might occur. The parallelism of hydration in biocolloids with hydration and growth in plants with regard to temperature implies identity of structural arrangement and similarity of action. The end points, maxima or totals in the swelling of the biocolloids given above probably represent the point at which dispersion assumes a high rate, and available information is not sufficient to identify this with the upper temperature limit of growth.

137 (1315)

The chemical basis of morphological polarity in regeneration

By JACQUES LOEB.

[*From the Rockefeller Institute for Medical Research.*]

When a piece is cut out of the stem of a plant (*Bryophyllum calycinum*) the most apical buds will grow out into shoots, while roots develop chiefly but not exclusively at the basal end. The writer suggests the following explanation of this phenomenon of polarity. In the normal stem the growing region at the apex as well as the leaves send out special inhibitory substances toward the base of the plant which prevent the growth of the more basally situated dormant buds capable of giving rise to shoots. When a piece is cut out from the stem these inhibitory substances continue to flow in the piece toward the base and the most apical node will be the first one sufficiently free from these inhibitory substances and hence the two dormant buds situated at this node will grow out first. As soon as they grow out they produce and also send out inhibitory substances toward the base, thereby preventing the more basally situated buds from growing out into shoots.

A brief outline of the experiments supporting this hypothesis has been published in *Science*.[2]

[1] Chemistry of Colloids, p. 59, 1917.

[2] Loeb, J., *Science*, 1917, xlvi, 547.

SCIENTIFIC PROCEEDINGS.

Abstracts of Communications.

Eighty-eighth meeting.

College of Physicians and Surgeons, January 16, 1918.

President Gies in the chair.

138 (1316)

Experimental studies of self-incompatibilities in fertilization.

By **A. B. Stout** (by invitation).

[*From the Botanical Gardens, Bronx Park, N. Y.*]

The phenomenon of self-incompatibility in the fertilization of hermaphrodites is well illustrated by conditions existing in the common chicory plant (*Cichorium Intybus*). The flowers are perfect, they are anatomically all alike, the flowers are open only for a short time so that pistils and stamens are ready for pollination at the same time, and the parts are decidedly adapted for self-pollination, and yet when controlled self-pollinations are made many plants, set no seed. Many cross-pollinations are also incompatible, but both pollen and pistils will function in certain crosses. The inability to set seed to self-pollination (or cross-pollination as well) is here best described as due to some sort of physiological incompatibility operating between sex organs (including sex cells themselves) that are fully formed, anatomically perfect, potentially functional and of simultaneous development.

Studies in chicory have been pursued by the writer during the past six years. Nearly 2,000 plants (all but the first crop were pedigreed) have been studied and controlled pollinations made of heads comprising a total of over 450,000 flowers. There is thus available more data on self-compatibility for this plant than for any other or perhaps for all other species in which the phenomenon has been studied. Publications giving data in detail and conclusions for results obtained previous to 1917 together with dis-

cussion of literature and theories have already been made. A brief summary of results and conclusions can be made here, however, as follows:

1. The results obtained with chicory indicate (*a*) that self- and cross-incompatibilities are strongly in evidence in this species; (*b*) that self-compatible plants may arise sporadically from parents that are self-sterile even after three generations of self-incompatible ancestry; (*c*) that the progeny of such plants do not breed true to this character; (*d*) that the degree of self-compatibility varies greatly; (*e*) that selection for high degrees of self-fertility continued for four generations has not been effective in isolating a completely self-fertile strain.

2. Self-compatibility and self-incompatibility are entirely independent of differences in vegetative vigor. In the selection experiments, races widely different in vegetative vigor have been isolated. Self-sterility and self-fertility appeared in all these races with much the same frequency. Sister plants of the least vigorous dwarf race or sister plants of the most vigorous vegetative race were either self-fertile or self-sterile indiscriminately.

3. Self-compatibility and self-incompatibility operate independently of potential sex vigor. The total production of flowers varied greatly among the various strains. Plants producing large numbers of sex organs were either self-sterile or self-fertile, as were plants with the fewest number.

4. Self-compatibility and self-incompatibility may operate independently of the purely nutritive relations of the embryos to their parent plants. Ten flower-heads self-pollinated on a completely self-sterile plant will set no seed, while ten heads on the same plant pollinated on the same day with pollen from a highly cross-compatible plant will set abundant seed. The fruits are rather small achenes having no endosperm and are practically composed only of the embryo: provided the pollination is compatible, they seem to develop equally well throughout the season.

5. Self-compatibility and self-incompatibility appear independently of any combination of germ-plasm elements in so far as these can be judged by the expression of characters. Each operates alike between gametes that are similar or those that are dissimilar in respect to hereditary units of genetic analysis. Plants

widely different in such qualities as color of flowers, type of branching, shape of leaves, etc., are either self-fertile or self-sterile, and plants of a sister series quite similar in all respects are either self-fertile or self-sterile. When an F_1 plant of hybrid origin is self-fertile in any degree, the evidence indicates that any of the sex cells may function in any recombination; on the other hand, in self-sterile sister plants whose sex cells must, it would seem, be of much the same diversity none are compatible. Also all the sex cells of an F_4 plant which must have much the same germ-plasm constitution may fail to function together, while those of a sister plant may be highly functional. Two self-sterile plants, sisters of an F_1 cross or sisters of any generation, may be cross-fertile or cross-sterile quite indiscriminately.

6. The development either of self-compatibility or of self-incompatibility occurs in both cross-bred and inbred races, the latter often being highly constant races. Both self-fertile and self-sterile plants occurred among sister plants that were F_1 hybrids of rather wide crosses; they also appeared among inbred strains derived by crossing self-sterile parents for as many as three generations, and they occurred among the progeny of self-fertile plants even after four generations of self-fertile parentage. The evidence at hand makes it clear that self-compatibilities do not necessarily decrease as a result of inbreeding.

7. The results obtained in the cultures of chicory make it clear that self-incompatibility and self-compatibility are here not to be described as dominant and recessive characters, or paired allelomorphs, and that there is no simple Mendelian formula that fits the results. The evidence at hand for the behavior of similar phenomena in other species is also quite in agreement with this conclusion.

8. The conditions controlling sex-fusions, judged by the behavior of compatibilities and incompatibilities in such species as *Cichorium Intybus*, arise in connection with the development of the sex organs and sex cells as such. In this sense the controlling factors are of epigenetic and individual development.

9. The factors which determine or prohibit successful fertilization in chicory, whatever their essential nature may be, are highly variable as to degree, specificity, and transmission in heredity.

10. In respect to the general results, the phenomenon of incompatibility appears to present some analogy to that of so-called antigen-antibody reactions in immunity, and to isoagglutination and isoprecipitation phenomena. Here, however, the incompatibility exists between organs produced side by side in the same flower and which are of close somatic relationship.

139 (1317)

A demonstration of cerebellar and cerebral lesions in dogs.

By I. STRAUSS and I. FRIESNER (by invitation).

[*From the Department of Physiology of Columbia University.*]

Dr. Pike with his characteristic modesty has asked us to present these dogs. They show lesions which resulted in part from design and in part from accident. The experiments were undertaken in an attempt to study the relations between the internal ear and the central nervous system.

From a clinical point of view otologists look upon the results of labyrinthine stimulation or destruction as divided into two heads:

1. Nystagmus.
2. Vertigo.

The brain pathways which intermediate the nystagmus impulses are well known. On the other hand, those which have to do with vertigo are very little known. Recently the theory has been advanced that the cerebellum intermediates the labyrinthine impulses which ultimately pass to the cerebrum, and have to do with our sense of position.

It was in the attempt to test this theory experimentally that these operations were undertaken. Some of the dogs show the result of injuries to the cerebellar peduncles. In another in attempting to reach the peduncles we accidentally injured the vermis and had to stop. Another dog shows the result of a bilateral cerebellar lesion as well as a vermis lesion. We have but commenced our studies and are indebted for our results largely to the kindly patience and help of Dr. Pike.

140 (1318)

Amylase and protease action of some pancreas preparations.

By H. C. SHERMAN and DORA E. NEUN.

[*From the Laboratory of Food Chemistry, Columbia University.*]

It was shown that the purified preparations of pancreatic amylase always exhibit a marked proteolytic activity whether tested by determination of total nitrogen of digestion products, by determination of amino nitrogen, by the Van Slyke method, by determination of acidity of digestion, or by the increase of electrical conduction.

While fractional precipitation by alcohol yields a first precipitate of increased proteolytic activity, the other (more soluble) fraction shows no increase in amylolytic power over the usual purified amylase preparation, and only a slight decrease of proteolytic power. The problem of the relationship of the amylolytic and proteolytic activities of these preparations was discussed.

The investigation was carried out with the aid of a grant from the Carnegie Institution of Washington.

141 (1319)

The rôle of afferent impulses in the control of respiratory movements.

By HELEN C. COOMBS and F. H. PIKE.

[*From the Department of Physiology of Columbia University.*]

As the result of further experimentation, we wish to add to a previous statement (*American Journal of Physiology*, 1917, XLII, p. 395) the following facts:

1. The movements of the ribs, which cease after division of the dorsal roots of the spinal nerves in the thoracic and cervical regions, are resumed when the phrenics are divided.

2. The respiratory movements become labored when the vagi are divided in the neck after these procedures, and soon cease. The effects of vagotomy are more severe if the dorsal roots of the

cervical nerves have been included in the section than if the thoracic nerves alone are affected.

3. The results of combined section of the dorsal roots of the spinal nerves and the vagi are similar to the effects of combined section of the brain stem below the corpora quadrigemina and the vagi. Transection below the corpora quadrigemina adds little or not at all to the severity of the effects following section of the dorsal roots.

4. Costal respiratory movements are resumed after section of dorsal roots, brain stem below the corpora quadrigemina and phrenics if the vagi are intact.

We believe that these experiments bring the intercostal muscles into line with the other skeletal muscles so far as the functional relations of afferent to efferent spinal nerve roots are concerned.[1]

We believe also that the experiments show that it is necessary that afferent impulses shall not merely have access to the central system, but that they shall go to a particular part of the central system in order to fulfill their function. In the case of the respiratory movements, afferent impulses from the intercostal muscles must go as far up as the corpora quadrigemina. The medulla oblongata seems sufficient for the establishment of any necessary connection of the vagi with efferent paths to the respiratory muscles.

142 (1320)

A case of hereditary ataxia (?) in pigeons.

By OSCAR RIDDLE.

[*From the Carnegie Station for Experimental Evolution, Cold Spring Harbor, L. I., N. Y.*]

From an egg produced under the weakening influences of "reproductive overwork" a female pigeon was hatched (in 1914) which showed a marked lack of power over the voluntary movements of the head and body. This lack of coördination was

[1] Sherrington, Schafer's "Text Book of Physiology," London, 1900, vol. ii, p. 797.

practically completely lost in the adult bird. The affected female was bred to two different males. The derangement has been inherited through four generations descended from either male.

The affected offspring have shown many degrees of the lack of muscular control (birds demonstrated). Some have shown marked disturbances when young and have later recovered. Others have at first been classed as normal and have later developed marked irregularity of movement. The more usual manifestations of the disorder are: Nodding of the head, or nodding and swaying of the head and neck; unsteady gait; tipping (somersaulting) backwards or forwards; falling on the side; very irregular flight, the bird even flying backwards. The same bird often exhibits two, three or perhaps all of these irregularities. Practically all affected birds are unable and uninclined to sit on a perch, remaining constantly on the ground, or on a flat ledge. In the most affected individuals there seem to be no movements whatever of wholly normal coördination; in average cases, however, the disturbances are much increased under excitement, fear, or any attempt at increased or more vigorous movement. In a few cases the movements have seemed fairly normal when the bird was at perfect rest. Several of the affected birds have mated and produced young. Offspring from two affected birds have not yet been obtained.

About 175 young have been reared to the age at which the disorder might be exhibited. Of this number 119 were classed as normal and 46 as affected. The first mating of the original female gave 33 normal and 3 affected. The affected offspring when mated to their unaffected relatives gave 11 normals to 14 affected. Similar affected individuals outcrossed have yielded 4 normals to 0 affected. There is some evidence that more of the affected individuals than normals die before attaining the age required for classification. The details bearing on the method of inheritance (chart demonstrated) make it appear that the new character is, with some irregularities, a Mendelian recessive.

The data are not sufficiently complete to permit a final statement concerning the inheritance of the character as limited by sex. That it is not wholly thus limited seems clear; possibly the sons of unaffected mothers exhibit the disorder more often than do

the daughters. The variation (mutation) has persisted through four generations.

143 (1321)

Chemical pneumonia.

By MARTHA WOLLSTEIN and S. J. MELTZER.

[*From the Laboratories of the Rockefeller Institute for Medical Research, New York.*]

The use of chloramine T solution in the treatment of inflammations of the nose and throat suggested to us that the solution might be useful in the curative treatment of pneumonia experimentally produced in dogs.

By intrabronchial insufflation of a definite dose per kilo of a virulent pneumococcus culture, it was possible to cause a pneumonia which proved fatal to dogs in about 36 hours. Insufflation of 5 c.c. per kilo of 1 : 10,000 solution of chloramine T in dogs previously insufflated with pneumococci brought out the fact that the treated dogs were harmed instead of benefited. The chloramine T was then used alone in normal dogs. In doses of 5 c.c. per kilo of a 1 : 10,000 solution it produced consolidation of the greater part of one or more lobes, with marked congestion and edema of both lungs. Microscopically the lesion was a broncho-pneumonia, with some intra-alveolar hemorrhage. Dakin's hypochlorite solution used in the same way caused a similar lesion, even in dilutions of 1 : 20,000. Bichloride of mercury in 1 : 10,000 solution produced rather more hemorrhage than the chlorine compounds did. Cultures made from the consolidated areas of all the lungs failed to grow. The pneumonia produced by these chemical substances was sterile.

144 (1322)

Changes in reflex thresholds following experimental shock from intestinal manipulation.

By EUGENE L. PORTER (by invitation).

[*From the Physiological Laboratory of the University of Pennsylvania.*]

Insensibility and apathy are common clinical observations in traumatic or surgical shock. Meltzer and others have reported similar observations when the shock is produced experimentally by withdrawing the intestines from the abdominal cavity and cooling or manipulating them. I have repeated this procedure with spinal cats and have subjected to quantitative measurement the effects produced on the cord. The cats are made spinal by pithing the brain through the foramen magnum. The flexion reflex is elicited by stimulation of the posterior tibial nerve and recorded by contractions of the tibialis anticus muscle. The Martin system of measuring the break shocks to the nerve is used. In such a preparation, left to itself at normal temperature, the threshold of the flexion reflex remains very constant for long periods of time, changing, if at all, very gradually. Ordinarily the changes between successive readings are fractions of a single unit of measurement. If, now, the intestines be withdrawn from the abdominal cavity and manipulated, in about 50 per cent. of the animals operated on, this threshold rises very promptly—within two or three minutes ordinarily—and within ten minutes it may have reached a value 50 per cent. greater than before manipulation. It commonly remains at this high level during manipulation. Upon cessation of the manipulation the threshold drops, and within ten minutes or less, it may be at its original level and continue at this level, showing as slight variations as before the manipulation. In such cases it may be possible to repeat the procedure on the same animal.

To take a specific case:

EXPERIMENT OF JULY 10, 1917.

3.13	Threshold	15.5	Z units.
4.36	Threshold	14.3	Z units.

(Frequent readings between 3.13 and 4.36. Change between successive readings never equal to a whole unit of stimulation.)

4.37 Intestines withdrawn and manipulated.

4.38 Threshold 16.8 Z units.

Threshold gradually rose.

5.07 Threshold 20.3 Z units.

5.08 Intestines returned.

5.16 Threshold 14.1 Z units.

6.00 Threshold 14.5 Z units.

The changes in threshold I have found to bear no relation to changes in blood pressure. The threshold may return to its original level with a lower blood pressure than at the time it went up.

The threshold of crossed-extension, obtained by stimulating the same nerve and recording the contractions of the quadriceps group of muscles, has a much more variable threshold than the flexion reflex before manipulation of the intestines, but quite evident changes in this threshold follow manipulation (Exp. of July 24). I have found the reflex in a number of cases to disappear for a few moments or longer as the result of the manipulation. The high threshold produced is less likely to return to its original level than the flexion threshold. The rise in threshold is just as prompt as in the case of flexion. In one case I have found a complete return to the original threshold, but only after an interval of an hour and a quarter after the manipulation of the intestines had ceased.

These results are of interest in connection with the present work on the shock problem. Pike has emphasized recently the probability of some physical injury to the central nervous system in shock. W. T. Porter has done the same in his theory of fat embolism. The injury I have studied is better described as physiological rather than physical. The prompt and complete return of threshold to its original value in some cases indicates that the injury cannot be so gross as the fat embolism of Porter. It is an inhibition, in the words of Meltzer, or an increase in synaptic resistance, as Cannon has recently referred to it.

The problem which immediately suggests itself is whether it

would be found possible to alter, by intestinal manipulation, the threshold of reflexes of more vital importance to the animal than flexion and crossed-extension. Porter has shown that the vaso-motor center is still in active operation in shock; it is by no means exhausted. It is still capable of giving as great percentile changes of blood pressure as before. It might still turn out to be true, however, that following intestinal manipulation the center is more inaccessible than before to the nerve impulses which ordinarily play upon it and properly control its activities. The writer hopes to test this out in the near future. At any rate it is evident that intestinal manipulation sends impulses to the central nervous system which disturb its normal functioning, and Crile's contention that such impulses should be prohibited from their pernicious activity would seem to be justified.

145 (1323)

On the influence of some antipyretics on the neuro-muscular coördination test of "tapping."

By DAVID I. MACHT, S. ISAACS and J. P. GREENBERG.

[*From the Pharmacological and Psychological Laboratories of Johns Hopkins University.*]

In connection with a psycho-pharmacological study of the antipyretics including observations on their effect on the reaction-time, blood pressure, vision, and hearing, some observations were made on the influence of these drugs on the well-known psychological coördination test of "tapping." This test consists briefly in the continuous tapping by the subject with a brass stylus upon a brass plate so adjusted that each tap or contact of the stylus on the brass plate is electrically registered on a counter. The number of taps made over a definite period of time is a rough index of the neuro-muscular coördination of the arm muscles. In the present investigation, observations were made upon the authors and occasionally on other subjects. The subject was required to tap continuously for three minutes at a time, and the number of taps registered was noted at the end of each minute. Having noted the normal tapping number in any one experiment,

the subject was given an antipyretic by mouth and the test was repeated, generally about an hour later, but in some cases several readings were repeatedly taken at definite intervals. The effect of the following drugs was studied: Phenacetin, antipyrin, acetanilid, quinine sulphate, pyramidon, aspirin and salol. In addition to the study of individual drugs, the following combinations were also administered: Acetanilid plus phenacetin, 5 grains each; phenacetin plus salol, $2\frac{1}{2}$ grains each and 5 grains each; aspirin and salol, 5 grains each; acetanilid and salol, $2\frac{1}{2}$ grains each and 5 grains each; and antipyrin and aspirin, 5 grains each. The results of the experiments were not very striking. Briefly, however, the effects of the drugs may be summarized as follows: Phenacetin, acetanilid, antipyrine and quinine in the ordinary doses (not exceeding 5 grains) showed a definite tendency to improve the tapping rate. Larger doses of these drugs (8 or more grains) tended to impair the efficiency of the test. The improvement after phenacetin and antipyrin was greater than that after acetanilid and quinine. After pyramidon, salol and aspirin, no definite change could be noted as the different subjects showed different results. Combinations of the various drugs studied all showed a tendency to improve the tapping rate. This was especially marked after the combinations of acetanilid and salol and phenacetin and salol.

SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

Eighty-ninth meeting.

College of the City of New York, February 20, 1918.

President Gies in the chair.

146 (1324)

On the relation of the chemical structure of the opium alkaloids to their effect on smooth muscle and on the discovery of a new therapeutic agent as a consequence thereof.

By DAVID I. MACHT.

[*From the Pharmacological Laboratory, Johns Hopkins University and the James Buchanan Brady Urological Institute, Baltimore.*]

In a paper dealing with the effect of opium alkaloids on the ureter,[1] the author has shown that the opium alkaloids in respect to their action on that organ can be divided sharply into two classes: the pyridin-phenanthrene group, of which morphin is the principal member on the one hand, and the benzyl-isoquinoline group, of which papaverin is the principal representative, on the other. The author showed that morphin and all its derivatives, with the exception of peronin or benzyl-morphin, all stimulate the contractions of the ureteral rings and increase their tonicity, while all the members of the papaverin group, namely, papaverin, narcotin and narcein inhibit the contractions of the ureter and lower its tonus.

In a subsequent paper[2] the author further analyzed the action of the opium alkaloids on the ureter and also studied a large number of other alkaloids and chemical compounds allied to them and was led to conclude that, as far as the stimulating action of the

[1] *Jour. Pharmacol. and Exp. Therap.*, 1916, IX, 197.

[2] *Jour. Pharmacol. and Exp. Therap.*, 1917, IX, 287.

morphin group on the ureter is concerned, that stimulating effect is to be ascribed to the *pyridin* part of the morphin molecule. Furthermore, the author showed that the inhibitory and tonus-lowering action of papaverin and its allied alkaloids appeared to be due to the presence of the *benzyl* grouping in the papaverin molecule. The skeleton structure of the morphin alkaloids and of the papevarin alkaloids is represented by the following structural formulæ:

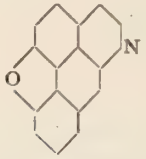


FIG. 1. Pyridin Phenanthrene Skeleton.

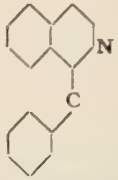


FIG. 2. Benzyl-Isoquinoline Skeleton.

It was further shown that when a combination of various opium alkaloids is used, the effect of the benzyl-isoquinolin members predominates in most cases. This explains also the anomalous effect of peronin or benzyl-morphin which, unlike all other morphin alkaloids, inhibits the contractions of the isolated ureter.[1]

Following the studies of the effect of the opium alkaloids upon the smooth muscle of the ureter, the author investigated the action of the same alkaloids upon other kinds of smooth muscle. The results of these investigations have in part been already published[2] and in part are to appear later. The smooth muscle structures which the author studied were the following: intestine, uterus, gall bladder, urinary bladder, biliary ducts, vas deferens, excised bronchioles, and arterial smooth muscle. As a result of these investigations, the author is led to conclude that the action of the opium alkaloids upon all smooth muscle is the same as that described in connection with the ureter, namely, that the morphin alkaloids *stimulate* contractions and increase tonus while the papaverin alkaloids *inhibit* contractions and lower tonicity, and furthermore, the experiments seemed to indicate especially in connection with the papaverin group that the inhibitory and tonus-

[1] *Jour. of Urology*, 1917, I, 201.

[2] *Jour. Pharmacol. and Exp. Therap.*, 1916, IX, 121; *Jour. Pharmacol. and Exp. Therap.*, 1917, IX, 473.

lowering action resides in the benzyl part of the papaverin molecule.

These observations and conclusions logically led the author to surmise that possibly the inhibitory and tonus-lowering effect of papaverin might be produced by the exhibition of a benzyl grouping in a simpler form. Accordingly a search was made for simple non-alkaloidal and non-narcotic compounds containing the benzyl radical which could be administered to animals without toxic results. Two such bodies were discovered. They were the well-known esters, *benzyl-benzoate* and *benzyl-acetate*. Inasmuch as these esters are practically insoluble in water, their pharmacological action could not be studied on excised tissues, but experiments on intact animals revealed the fact that both of these esters actually produced the same pharmacological effects as those given by the opium alkaloid papaverin, on the one hand, and that these esters were much less toxic than papaverin, on the other. Metabolic studies in connection with these experiments showed that the benzyl group of benzyl-benzoate and benzyl-acetate is in a large measure converted into hippuric acid in the body and is excreted as such in the urine. The comparatively very low toxicity of benzyl-benzoate and benzyl-acetate for animals and their striking papaverin-like effects on various viscera led the author to try their action upon himself and as no untoward or even disagreeable symptoms followed the ingestion of these drugs by mouth, it was deemed justifiable to administer these agents to suitable patients with their consent.

The conditions in which the benzyl effect was anticipated to produce pharmacological result, were those exhibiting either *excessive peristalsis* or excessive *spasm* of smooth muscle. It would take too much space in this preliminary communication to describe in detail all the cases in which benzyl-benzoate for the most part and benzyl-acetate to a lesser extent, were tried. The author need only state in this place that the oral administration of these esters was followed by remarkable pharmacological and therapeutic results of a beneficial character in the following conditions: (1) Excessive peristalsis and colic of the intestines, namely, in cases of diarrhea and dysentery. (2) Spasm and colic of the ureteral smooth muscle, that is, "renal colic." (3) In

"biliary colic " or spasmodic contractions of the gall bladder. (4) In uterine colic or spasmodic contractions of the uterus. (5) In vesical colic or spasmodic contractions of the urinary bladder. (6) In one case of spastic constipation with powerful tonic spastic contraction of the intestine. (7) In a few cases of pylorospasm. (8) In a large number of cases of arterial spasm or hyper-tension. (9) Lastly, one of the most striking of all the effects, in cases of bronchial spasm or true asthma. Administration of these drugs by injection has also been tried. A complete pharmacological study together with further therapeutic observations on the action of the above benzyl-esters and benzyl-alcohol will be published in due time in the Journal of Pharmacology and Experimental Therapeutics. This preliminary announcement, however, is made in this place because it is deemed that a sufficient number of observations, both pharmacological and clinical, have already been recorded by the author and a number of physicians who have kindly collaborated with him, to indicate that the benzyl-esters promise to become useful therapeutic agents.

147 (1325)

Studies in calcium and magnesium metabolism. Further observations on the effect of acid and dietary factors.

By MAURICE H. GIVENS.

[*From the Sheffield Laboratory of Physiological Chemistry, Yale University, New Haven.*]

Givens and Mendel[1] have found that "Administration of hydrochloric acid produced no significant effect upon the balance of N, Ca, and Mg in the dog." Stehle[2] has stated that "the administration of hydrochloric acid by mouth to the dog causes an increased excretion of calcium and magnesium as well as of sodium and potassium." New experiments have been conducted on two dogs which received a diet poor in lime, consisting of meat, cracker meal, lard, and agar. During two long periods each animal received daily 2 gm. hydrochloric acid. The results with

[1] Givens, M. H., and Mendel, L. B., *J. Biol. Chem.*, 1917, XXXI, 421.

[2] Stehle, R. L., *J. Biol. Chem.*, 1917, XXXI, 461.

such a diet corroborate our former statement and do not support the conclusion of Stehle.

Similar experiments were undertaken with these dogs on a diet enriched in lime by the addition of dried milk. With one animal a negative calcium balance was appreciably increased. It seems unlikely, however, that this outcome is a neutralization phenomenon. It has repeatedly been shown that when acid is introduced into the organism the increased production of ammonia is sufficient to neutralize it.

When sodium chloride instead of hydrochloric acid was added to the diet rich in lime the urinary calcium output was increased and the calcium balance was favorably affected.

The magnesium balance and the partition of this element between the different paths of excretion was not significantly altered in any of the experiments just described.

148 (1326)

Studies on salt action. I. Effect of calcium and sodium salts upon the viability of the colon bacillus in water.

By **C.-E. A. WINSLOW** and **I. S. FALK.**

[*From the Yale School of Medicine.*]

The experiments here reported are preliminary observations designed to test the suitability of a certain bacterial characteristic, the curve of viability in water, for use in more extensive studies of salt action planned for the future.

The organisms used were typical colon bacilli of the *B. communis* (sucrose negative), type, isolated from a polluted stream in the autumn of 1916. These bacteria were grown on standard nutrient agar slants at 37° C. for 16–18 hours. The growth from the surface was washed off in pure water, shaken for 5 minutes to break up clumps and added in 1 c.c. portions to the bottles of sterilized pure water or salt solution in which the viability was to be tested. These solutions had been warmed to 37° before seeding. Plates were made on agar one minute after seeding and the bottles were then replaced in the 37° incubator and kept there for 24–52 hours, the number of surviving organisms being determined

at stated intervals. Counts were made on standard agar after incubation for 24 hours at 37° C.

The "pure water" used was twice redistilled in glass vessels and again redistilled in a block tin condenser. It gave a negative test for ammonia with Nessler's reagent. The sodium chloride was purified by treatment with calcium hydroxide (to remove magnesium), and with sodium carbonate (to remove calcium and barium), and was then recrystallized from pure hydrochloric acid and from pure water. The calcium chloride was twice recrystallized and the crystals dried in a current of air and in the drying oven.

TABLE I.

EFFECT OF CALCIUM ALONE.

Sol.	Grams $CaCl_2$ in 100 C.c. Water.	Theoretical Tonicity.	Bacteria per C.c. Time after Seeding.				
			1 Min.	3 Hrs.	6 Hrs.	9 Hrs.	24 Hrs.
A ..	0.000		24,400,000	36,700,000	34,900,000	34,200,000	17,200,000
B ..	0.161	0.1 iso	21,100,000	13,100,000	4,900,000	4,800,000	2,900,000
C ...	0.805	0.5 "	27,900,000	2,100,000	230,000	140,000	44,000
D ..	1.610	1.0 "	18,200,000	2,800,000	160,000	53,000	170
E ..	8.050	5.0 "	11,400,000	14,500	0	0	0

TABLE II.

EFFECT OF SODIUM ALONE.

Sol.	Grams NaCl in 100 C.c. Water.	Theoretical Tonicity.	Bacteria per C.c. Time after Seeding.				
			1 Min.	2 Hrs.	6 Hrs.	25 Hrs.	52 Hrs.
A ..			25,130,000	25,030,000	23,650,000	23,500,000	20,000,000
B ..	0.085	0.1 iso	40,230,000	39,250,000	46,080,000	42,500,000	40,000,000
C ...	0.425	0.5 "	40,650,000	36,530,000	48,600,000	25,500,000	25,100,000
D ..	0.850	1.0 "	34,350,000	43,300,000	41,430,000	38,000,000	16,850,000
E ..	4.250	5.0 "	2,420,000	163,000	5,000	900	750
F ...	8.500	10.0 "	4,540,000	42,500	0	0	0

TABLE III.

EFFECT OF CALCIUM AND SODIUM TOGETHER

Sol.	In 100 C.c. Water, Grams.		Theoretical Tonicity.	Bacteria per C.c. Time after Seeding.				
	NaCl.	$CaCl_2$.		1 Min.	3 Hrs.	6 Hrs.	9 Hrs.	24 Hrs.
A ..	.355	.134	0.5 iso	1,940,000	2,150,000	2,800,000	5,200,000	7,600,000
B ..	.710	.268	1.0 "	1,050,000	3,850,000	2,500,000	4,900,000	4,800,000
C ..	1.420	.536	2.0 "	910,000	3,200,000	4,300,000	4,400,000	4,600,000
D ..	3.550	1.340	5.0 "	1,020,000	1,275,000	1,200,000	2,200,000	1,260,000
E ..	7.100	2.680	10.0 "	890,000	98,000	34,000	45,000	8,000

Ratio of Na ⟷ Ca is the same in all solutions (5 ⟷ 1).

The solutions to which the bacteria were exposed contained respectively, calcium chloride alone, sodium chloride alone, and a mixture of the two salts in the ratio of five molecules of sodium to one modecule of calcium. The tonicity varied from nothing to a ten times isotonic condition.

It is evident that the calcium salt alone exerts a marked lethal action. Even a 0.1 isotonic solution shows a distinct reduction in bacterial numbers and increasing proportions grow more deadly till a 5.0 times isotonic solution produces sterility in 6 hours. The sodium salt is less harmful. Tonicities of 0.5 and 1.0 show a slight effect, and a tonicity of 5.0 a marked effect, but the latter concentration does not completely sterilize even after 52 hours. A mixture of the two salts in proportion of 5 parts of sodium to one of calcium is much more favorable than either salt alone. Up to and including a tonicity of 5.0 the bacteria actually increase in numbers. Line *D* of Table III shows that 3.5 per cent. sodium chloride plus 1.3 per cent. calcium chloride had no harmful influence, while in Table II .4 per cent. of sodium chloride alone, and in Table I less than .2 per cent. of calcium chloride alone exerted a demonstrable lethal effect.

The toxic effect of calcium and sodium salts and their antagonistic influence upon each other seem to be much the same among the bacteria as in the higher forms of life; and the viability curve in water would seem well suited for the study of such phenomena.

149 (1327)

Protein sparing by glucose in experimental diabetes.

By **N. W. JANNEY** and **V. I. ISAACSON.**

[*From the Montefiore Home and Hospital, New York City.*]

In diabetes there are abnormalities not only in carbohydrate but also in protein and fat metabolism as well. There is frequently a marked loss of protein in this disease, which may be either direct or the result of a failure on the part of carbohydrate to spare protein. Our experiments deal with one phase of protein metabolism in experimental diabetes, the influence of the removal of the major portion of the pancreas on the characteristic protein sparing action of glucose. For this purpose several female dogs

weighing between nine and fifteen kilograms each were selected. Both the fasting nitrogen elimination and the effect of glucose feeding on this excretion were determined. During the fasting period the animals received only 40 c.c. of water per kilo. On the sugar days they received in addition 15 gm. of glucose per kilo. An after-period during which the glucose feeding was withdrawn was then introduced. Dr. Allen, of the Rockefeller Institute, then removed surgically about 90 per cent. of the pancreas. The wounds healed aseptically and the animals were allowed from two to five weeks to recuperate. The procedure consisting of fore-glucose and after-periods just described was repeated. In spite of the large loss of pancreatic tissue the protein-sparing action of glucose was not diminished in any of the animals. It appears that pancreatic tissue is provided far in excess of actual requirements or that this gland is not solely responsible for the protein-sparing action of carbohydrates. Dr. Ringer found this function intact in dogs made completely diabetic by means of phlorizin.

150 (1328)

Studies in experimental shock.

By **T. S. GITHENS** and **S. J. MELTZER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

The experiments were made on etherized dogs. The intestines and stomach were dislocated and frequently handled. Two manifestations were studied, the blood pressure and the pain sense. We shall state merely the facts obtained.

About one third of the dogs were given a preliminary injection of morphin. Some of the animals were given ether by cone, others by the method of intratracheal insufflation. These factors had no apparent influence on the results.

There were 42 dogs which received surgical narcosis. In 15 the blood pressure never reached a level below 95 mm. even when the experiment lasted 4 hours or longer, and sensation returned as soon as the animals came out of ether. In 13 dogs the blood pressure reached a level below 70 mm. of mercury within two and

a half hours and there was no return of pain sense. In 8 dogs the blood pressure sank to a low level but sensation of pain returned when ether was discontinued. In the remaining 6 dogs sensation was lost while the pressure was still above 95 mm. of mercury.

In 10 other dogs etherization was pushed to the point of intoxication. Interpretation of the results of this series is not simple, but it may be stated that the blood pressure, which fell in some instances to 42 mm. often continued to rise for as much as two hours after stopping the ether, eventually reaching a level above 90 mm. Pain sense sometimes returned after an even longer interval, but eventually recovered more completely than the blood pressure.

151 (1329)

Further observations on the nutritive factors in animal tissues.

By LAFAYETTE B. MENDEL and THOMAS B. OSBORNE.

[*From the Sheffield Laboratory of Physiological Chemistry in Yale University, and the Laboratory of the Connecticut Agricultural Experiment Station, New Haven.*]

A recent communication[1] reported that the proteins of meat (dried skeletal muscle) and of a glandular tissue, liver, are adequate for the needs of nutrition in growth. The muscle contains a comparatively small quantity only of the water-soluble vitamine that is an essential dietary factor, whereas liver tissue, on the other hand, was found to be much richer in this. We have further observed a similar adequacy of the proteins of heart, kidney and brain tissue. The growth of rats to adult size has been secured upon diets in which these tissues, used desiccated, furnished the sole source of protein and water-soluble vitamine. In the cases of the liver, kidney and heart, at least, fat-soluble vitamine also seems to be present. The ether extract of pig's liver—liver oil—has manifested growth-promoting properties comparable to those described for butter fat and cod-liver oil; and it appears to be efficient as a curative agent for the type of eye disease which we

[1] Osborne, T. B., and Mendel, L. B., "Nutritive Factors in Animal Tissues," I, *Jour. Biol. Chem.*, 1917, XXXII, 309.

have described to occur when animals are kept on a diet deficient in the fat-soluble vitamine. The details will be published in the Journal of Biological Chemistry.

152 (1330)

The effect of continued inbreeding on the tumor rate in mice.

By LEO LOEB and A. E. C. LATHROP.

[*From the Department of Comparative Pathology of Washington University Medical School, St. Louis, Mo.*]

In former investigations[1] we found that different strains of mice differ very much in their tumor rate and tumor age; and that these differences in tumor rate and tumor age were maintained and hereditarily transmitted to the following generations throughout the period of observation, notwithstanding the similarity of external conditions under which the animals were kept.

It was of interest to inquire how continued inbreeding of these various strains would affect the tumor rate and tumor age.

We found that while in the majority of strains the differences in tumor rate and tumor age remained constant in the successive generations, in others certain variations became noticeable as a result of the continued propagation of these strains. In a few cases an increase in the tumor rate occurred in the later generations, but in the large majority of cases in which a change was observed there was a tendency towards a decrease in the tumor rate which in some instances was very marked.

These changes in the tumor rate concomitant with continued inbreeding seem to depend on the following two factors: (1) As a result of long-continued inbreeding in mice certain characteristics of a strain may change, the strain becoming less prolific and less vigorous. These changes may be accompanied by a lowering in the tumor rate. This was especially evident in the case of the "No. 8" strain which had been inbred through the largest number of generations and through a considerable number of years. (2) In other cases it could be shown that in the course of continued

[1] A. E. C. Lathrop and Leo Loeb, *Proceed. Soc. Biol. and Medic.*, 1913, XI, 34; *Journal of Exper. Medicine*, 1915, XXII, 646, XXIII, 713.

propagation certain families, which were more resistant to certain diseases or which were naturally more prolific or otherwise favored by accidental conditions began numerically to preponderate in later generations. In some cases such families or substrains differed in their tumor rate from the main strain and thus as a result of selection within a strain differences in the tumor rate appeared in the course of continued inbreeding.

Our further investigations confirmed and still further emphasized our previous conclusion that on the whole in the mice with a higher tumor rate the tumors appear at a relatively earlier age than in those strains in which the tumor rate is lower.

153 (1331)

Sulpho-conjugation as a test of hepatic function.

By MAX KAHN.

[*From the Department of Laboratories, Beth Israel Hospital, New York.*]

It was demonstrated by Baumann and others that the toxic carbocyclic radicals split off from the proteins by the growth of intestinal bacteria are conjugated in the liver with sulphuric acid and thus detoxicated. It is also known that a portion of these aryl compounds are detoxicated by conjugation with glycuronic acid.

It is obvious that in testing for the functional capacity of the liver it is essential not only to test the glycogenic, ureogenic, biligenic etc., functions, but also to examine the detoxicating power of the hepatic tissue, in order to ascertain the complete working power of the gland. This is done in the following manner: The patient receives a dose of castor oil to evacuate his bowels. He is then kept on a known diet for two days, during which time the urine is collected, preserved and analyzed for total sulphur and ethereal sulphates. On the third day the patient receives a capsule containing 0.5 gm. thymol. The urine is collected for the next two days, preserved, and again analyzed for total sulphur and for ethereal sulphates.

If all the thymol were absorbed, and if all the thymol were

conjugated with sulphuric acid and none with glycuronic acid, the 0.5 gm. thymol would be excreted as 0.766 gm. of thymol sulphuric acid. This would cause a marked increase in the percentage of ethereal sulphates in the urine. If the detoxicating power of the liver were below par, the thymol would not be conjugated, and the percentage of ethereal sulphates would be only slightly different from what it had been on the first two days—before the thymol administration.

We have found that this detoxicating function of the liver usually runs parallel with the other functional derangements of this organ. In some cases, however, the conjugating power of the gland is markedly reduced, whereas the other functions do not show any disturbances as determined by the methods at our disposal. In still other cases the sulpho-conjugation is entirely normal (as determined by the test here described), while the other tests show a reduction of hepatic functional capacity.

It is possible that each individual cell of the liver tissue takes part in all of the liver functions; it is also possible that different portions of the liver lobule, and different conglomerations of liver lobules may have specific functions. In the former case, it is most likely that a reduction in the ability of the liver to perform one function will be accompanied by a proportional reduction in all the liver functions; in the latter case one or more functions of the liver may be disturbed without affecting the other hepatic functions.

It is essential, in studying hepatic disease to examine all the functions of the liver by the various methods at our disposal, and it is important to examine the detoxicating power of the liver before drawing any conclusions as to the type and extent of hepatic involvement.

154 (1332)

The relation of the intestinal flora to the scurvy of guinea pigs and of infants.

By J. C. TORREY and ALFRED F. HESS.

[*From the Department of Health, New York City.*]

The question recently has been raised as to whether scurvy in guinea pigs and in human beings is due to constipation and to the putrefactive activity of the bacteria in the intestinal tract. In

order to determine this point a study of the intestinal flora was made in guinea pigs on a normal diet, on a diet which produced scurvy, and again on a diet which cured this disorder. For this purpose cultures and grain stained films were made from the feces, as well as from the different levels of the small and large intestine immediately after chloroforming the animals. This study on guinea pigs is portrayed in Table 1.

It will be seen that proteolytic bacteria other than subtloid types were not found in the intestinal tract; that the bacteria which were cultivated were merely those found in the outer world, for example, on dried foodstuffs. Similar organisms, indeed, were cultivated from the hay and oats which were fed these animals. Attention may be called to the fact that with none of the diets did *B. coli* develop, and that it was isolated from only one animal. The number of viable bacteria will also be noted as remarkably few, generally less than 1,000 per milligram of material. Among these there were hardly any that were actively proteolytic. Furthermore, a point of prime importance, there was no change in the flora on adding orange juice to the diet, although the scorbutic symptoms disappeared. It is our opinion, therefore, that most of the bacteria entering the intestinal tract are destroyed, and that the pabulum is not such as to encourage the growth of native intestinal bacteria.

A similar study was carried out on the stools of three infants suffering from scurvy. Table II gives a summary of these results. It will be seen that the infants were all on a high carbohydrate diet, and that in two instances we were able to compare the flora, not only during the active scorbutic process, but after orange juice had been given for a week or more. This investigation shows that the bacteria were merely such as we should expect with a diet rich in carbohydrates. Putrefactive organisms were present only in small numbers; in the case where they were found to be most numerous (*S*) they had disappeared upon the subsequent examination, although the scurvy had become more marked.

We therefore conclude from this study that the scurvy, both of guinea pigs and of infants, is not associated with an overgrowth of putrefactive bacteria in the intestinal tract. The amount of putrefaction present, however, can be determined only by a chemical study.

TABLE I.

INTESTINAL FLORA OF NORMAL AND OF SCORBUTIC GUINEA PIGS.

Guinea Pig.	Diet.	Period of Feeding.	Scorbutic Condition.	Source of Material Cultured.	Types of Bacteria.	Remarks.
No. 1664	Straw, oatmeal, water			Cecum	Staph. alb. and aur. Subtiloid Gram—bac. Yeasts	No B. coli Very few viable bacteria
		20 days	Scurvy	Colon	Subtiloid Gram +diploc. B. acidoph. Yeasts	No B. coli Material almost sterile
				Ileum	Staphyloc. Gram + diploc. Yeasts	No B. coli No putrefactive types About 1 viable organism per mg.
H. 63	Straw, oats, water	19 days	Scurvy	Cecum	Subtiloid bac. Gram + cocci B. acidoph. Yeasts	About 1 viable organism per mg.
				Colon	Like cecum	Like cecum
No. 414	*Carrots*, hay, water	Several weeks	Normal	Feces	Subtiloid bac. B. acidoph. Gram + diploc. Yeasts	No B. coli (or other gas-producing bact.) 20,000 viable organisms per mg.
	Oats, hay, water	8 days	Normal	do.	Flora unchanged	No B. coli 1,000 viable organisms per mg.
	do.	15 days	Scurvy	do.	Gram—bac. (saprophytic) Staph. alb.	No B. coli 16,000 viable organisms per mg.

TABLE I.—*Continued.*

Guinea Pig.	Diet.	Period of Feeding.	Scorbutic Condition.	Source of Material Cultured.	Types of Bacteria.	Remarks.
	Oats, hay, *orange juice*, water	3 days	Normal	do.	Subtiloid bac. (spore bearing) Gram + diploc. Yeasts	No B. coli 56 viable organisms per mg.
	Do.	8 days	Normal	Ileum	Streptoc. Gram + cocci B. acidoph. Yeasts	No B. coli 1,700 viable organsims per mg.
				Cecum	Moulds Yeasts Sporebearing bac. (numerous) M. ovalis	No B. coli 640 viable organisms per mg. putrefactive tendency
				Rectum	Yeasts Streptoc. B. acidoph.	No B. coli 840 viable organisms per mg.
				Ileum	Yeasts B. acidoph. Saprophytic bact.	No B. coli No proteolytic bact. 264 viable organisms
No. 1776	Raw goat's milk, hay	87 days	Scurvy	Cecum	Yeasts B. acidoph. Gram—twisted bac. M. ovalis	No B. coli No proteolytic bact. 560 viable organisms
				Rectum	Yeasts B. acidoph. Saprophytic bact.	No B. coli No proteolytic bact. 4,380 viable organisms

TABLE II.

FECAL FLORA OF SCORBUTIC INFANTS.

Infant.	Diet.	Date.	Scorbutic Condition.	Source of Material.	Types of Bacteria.	Remarks.
M.	Malt soup and cereal	Dec. 1	Subacute	Rectum	B. acidoph. B. bifidus M. ovalis B. coli	Normal infants' flora. Gram + bac. predominant No spore-bearing or putrefactive types B. acidoph. about 40% viable bact.
	Do.	Dec. 4	Do.	Do.	Do.	Do.
	Same also, 20 c.c. liquid petrolatum, one week	Dec. 11	Subperiosteal hemorrhage	Do.	Do.	Relatively more B. coli Many B. bifidus No putrefactive bact.
	Same diet, oil stopped, *orange juice*, 10 days	Dec. 21	Markedly improved	Do.	Do.	Normal infant's flora Bacteria as above
K.	Malt soup and cereal	Dec. 11	Subperiosteal hemorrhage	Rectum	Streptococci B. coli M. ovalis	Gram—bacteria predominant B. coli gram + diploc. numerous B. acidoph. few B. bifidus few
	Do. + *orange juice* (60 c.c.), 8 days	Dec. 21	Markedly improved	Do.	B. bifidus B. coli Streptococci	Gram + bact. predominant Many B. bifidus Streptoc. unchanged
S.	Formula: Cream, water, flour, sugar, also cereal	Dec. 21	Mild scurvy	Rectum	B. lact. aerog. B. coli M. ovalis B. bifidus B. welchii	Gram + and—bact. about equal Many lact. aerog. Putrefactive bact. in minority
	Malt soup and cereal	Feb. 11	More marked	Do.	B. lact. aerog. M. ovalis B. bifidus Dipheroids	Gram + bact. in great majority (B. bifidus) Spore bearers very few Flora not at all putrefactive

Abstracts of the Communications,

Pacific Coast Branch.

Nineteenth Meeting.

San Francisco, California, February 6, 1918.

155 (1333)

Sources of error in the estimation of dextrose by the colorimetric picrate method.

By **T. Addis** and **A. E. Shevky.**

[*From the Laboratory of the Medical Division of Stanford University Medical School, San Francisco.*]

A study has been made of the factors involved in the development of the red-brown color produced on heating solutions containing dextrose, picric acid and sodium carbonate.

The dextrose concentrations chosen corresponded in range to those found in the blood. Temperature levels above 100° C. were attained by heating in an autoclave, and the required accuracy of temperature control was obtained by means of an electric thermo-regulator.

The amount of color produced did not vary directly with the amount of sugar present except at a certain definite concentration of sodium carbonate. At low alkali concentrations small amounts of sugar gave relatively less color than large amounts, while at high alkaline concentrations, the reverse was found. The optimum alkali concentration varied with temperature changes.

With every increase in temperature from 80 to 130 ° C. there was an increase in color production, and at all temperatures the more prolonged the heating, the deeper was the color.

156 (1334)

Does the liver secrete a catalase accelerator?

By THEODORE C. BURNETT.

[*From the Rudolph Spreckels Physiological Laboratory of the University of California.*]

All observers are agreed that the catalytic activity of the liver is far in excess of that of muscle, and it is generally assumed, I think, that this is because the liver contains more catalase. Assuming the liver to be five times more active than muscle, a mixture of muscle and liver in the proportion of five to one ought to give the same catalytic activity as double the quantity of muscle alone. This is not the case, as the following experiment shows.

A 50 per cent. solution of commercial hydrogen peroxide, representing 1.5 per cent. H_2O_2, was used. The oxygen was collected over water in the usual way, and the volume reduced to 0° and 760 mm. Hg. The time was 10 minutes.

1 gm. rat's muscle gave 50 c.c. oxygen.
1 gm. rat's liver gave 244 c.c. oxygen.
0.5 gm. muscle + 0.1 gm. liver gave 250 c.c. oxygen.

To be on the safe side, the assumption has here been made that the liver is five times as active as the muscle, yet the mixture, instead of having the activity of the muscle, has the activity of the liver.

Assuming the liver to be ten times as active as muscle, the following combination was made: 0.5 gm. muscle + 0.1 gm. liver. This combination gave 224 c.c. of oxygen.

Blood clot + muscle gives similar results, and to a less extent, spleen also. The difficulty of getting spleen free from blood complicates the matter.

The above facts appear to be certain. The interpretation is not so clear, but it may be the liver secretes an accelerator of catalase, perhaps in the nature of an internal secretion; and it may be it is this accelerator that causes the catalase activity to vary in the different tissues and organs.

SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

Ninetieth meeting.

Presbyterian Hospital, March 20, 1918.

President Gies in the chair.

157 (1335)

The rate of dialysis of diabetic blood-sugar.

By ISRAEL S. KLEINER.

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

Fifteen or twenty c.c. of blood, rendered incoagulable by dry hirudin, were dialyzed in a very thin animal parchment bag against 1,000 c.c. of Ringer's solution. To the Ringer's solution was sometimes added a small percentage of dextrose in order to retard the rate of dialysis. Bacterial growth was inhibited by the addition of powdered thymol or by working at a low temperature. Samples of blood for sugar analysis were taken at hourly intervals and the rate at which the sugar passed from the blood into the Ringer's solution was thus determined.

The results show a striking difference between the diabetic blood (from depancreatized dogs) and its control, normal dog blood with enough added dextrose to give about the same percentage of sugar as the diabetic. In the control a smooth curve is obtained, *i. e.*, the most rapid dialysis during the first hour, somewhat less the second, and so on. The diabetic, however, exhibits a marked slowing, or even a complete cessation, of dialysis usually during the second hour, but at times this occurs at some other period and there may even be two such periods in the course of a four-hour dialysis.

This may indicate that at least part of the diabetic blood-sugar is in a difficultly diffusible state, perhaps in some loose combination.

158 (1336)

Experiments on the scurvy of guinea pigs.

By ALFRED F. HESS and LESTER J. UNGER.

[*From the Bureau of Laboratories, Department of Health.*]

The diet used consisted of whole oats, hay, and water. Hay was added in order to supply the ash of green fodder; it was found, however, to have additional value owing to its roughage, and not to be replaceable in the dietary by its ash. In some instances cod-liver oil was added to this diet, without prolonging the life of the pigs or manifestly improving their condition. This diet has the advantage of bringing about scurvy in all animals without exception, and in leading to scorbutic signs in pigs weighing 200 to 300 grams in a period of two to three weeks. It thus permits of a quantitative estimation of the influence of diet, or other factors, in relation to scurvy. The addition of orange juice to this dietary leads to a marked and long-continued gain in weight, and the disappearance of all scorbutic manifestations.

In carrying out experiments of this kind it is well to chart the progress of the scurvy, as well as the growth in weight, as it is quite possible for the weight to decrease, although the scurvy is undergoing cure. For example, if straw is substituted for the hay in this dietary this twofold phenomenon takes place. An empirical method of graphically charting the course of scurvy is suggested.

The pigs fed on oats, hay and water were not constipated, as they frequently are when given a milk diet; nor was the cecum of those dying of scurvy found to be impacted, but contained gas and semi-solid feces. An addition of albolene, in daily dose of 0.5 to 1 c.c., did not serve either to protect or to cure the animals.

159 (1337)

Types of pneumococci found in the mouths of surgical cases before operation.

By MIRIAM OLMSTEAD.

[*From the Bacteriological Laboratory, The Presbyterian Hospital.*]

An investigation of post-operative pneumonia during 1916 and 1917, a report of which has been made by Dr. Whipple,[1] involved a routine search for pneumococci in the mouths of cases admitted to the surgical service of The Presbyterian Hospital.

The method of procedure was as follows: A specimen of sputum or saliva, whatever the patient could expectorate, was received in a sterile jar and sent to the laboratory. The specimen was injected into a mouse and the type of pneumococcus recovered from the mouse was determined by agglutination reactions, testing a pure culture against pneumococcus serum furnished by courtesy of The Rockefeller Institute. When a patient could not expectorate, the throat was swabbed, a culture made from the swab and pneumococci isolated from the culture. After a few months, only expectorated specimens were used, owing to the small percentage of cultures yielding pneumococci. From February to June, 1916, two hundred and forty-nine swab cultures were examined, from forty-three of which pneumococcus was recovered, a percentage of only 17.2. The incidence of pneumococcus in four hundred and twenty-nine expectorated specimens during the same period was one hundred and seventy-five, a percentage of 40.7. If it had been feasible to inoculate a mouse with the washings from a swab immediately after its leaving the mouth, this method of detecting the presence of pneumococcus in the mouth would have been satisfactory; but in consideration of the technique employed it is highly probable that 17.2 per cent. underestimates the number of individuals in that group harboring pneumococci. However, in tabulating the results according to type, these individuals have been included.

Two thousand four hundred and seventy-seven specimens have been examined, two thousand two hundred and twenty-eight

[1] Whipple, A. O., *Surg. Gyn. and Obstet.*, 1918, XXVI, 29.

of them expectorated specimens, two hundred and forty-nine swabs. Pneumococcus was recovered from seven hundred and ninety-eight specimens, 32.2 per cent. If the unsatisfactory swab group is subtracted, the percentage of positive results for two years is 33.8. In 1916 the annual percentage was 36.4, in 1917, 32.5. The lowest percentage at any time was 20, in November, 1917; the highest was 49, in March, 1916. During both years the percentage was comparatively high from February through June. Eighty-seven out of the two thousand four hundred and seventy-seven cases studied developed post-operative pneumonia. Pneumococcus was recovered from the pre-operative sputum of forty-seven of these eighty-seven cases, a percentage of 54.

The table summarizes the findings according to type.

	Incidence.	Per Cent.
Type I	5	.6
Type II	10	1.2
Atypical II	90	11.2
Type III	104	13
Type IV	589	73.8
Pneumococcus present	798	32.2
Pneumococcus absent	1,679	
Total cases	2,477	

Some of the Type IV. pneumococci have been classified and all of our twelve groups are represented among them.[2]

A history of contact with a pneumonia case was obtained in only one instance, that of an individual with Type I in the sputum. This was the only member of the Type I group that developed post-operative pneumonia, forming 20 per cent. One with Type II developed post-operative pneumonia, 10 per cent., seven with an Atypical II, 7.7 per cent., nine with Type III, 8.6 per cent., twenty-nine with Type IV, 4.9 per cent. There seems to be no predominance of the virulent types at any one period of the year. Among the Type IV strains, there was great variation in virulence, mice succumbing in from eighteen hours to eight days after inoculation of sputum, the majority within three days.

Dochez and Avery[3] reported finding the pneumococcus in 58.4 per cent. of the mouths of one hundred and thirteen "normal

[2] Olmstead, M. P., *Jour. Immunol.*, 1917, II, 425.

[3] Dochez and Avery, *Jour. Exp. Med.*, 1915, XXII, 105.

individuals." The high percentage may be partly accounted for by the fact that eleven of these had been or were in contact with pneumonia cases of Types I and II. Stillman[1] reported a series of three hundred and ninety-eight "normal individuals," finding pneumococcus in one hundred and seventy-two (43.2 per cent.). The specimens examined by us were not, strictly speaking, from normal mouths; but inasmuch as these cases were in the hospital because of conditions unassociated with respiratory affections, it seems quite fair to compare the results with those reported by Dochez, Avery and Stillman. Stillman's figures contain a greater proportion of Types I, II and III than do ours, but most of the individuals harboring Types I and II were "contacts." In our series, only one of whom was known to be a "contact," the virulent types were relatively uncommon, 73.8 per cent. of the cases harboring Type IV pneumococci. There is apparently a considerable variation in the incidence of pneumococci in the mouths of normal individuals at different seasons, in different years and in different groups, and among the individuals harboring pneumococci considerable variation in the proportion of the four types.

Summary.—During 1916 and 1917 pneumococci were recovered from the mouths of 32.2 per cent. of surgical cases before operation. Type I was found in .6 per cent. of the cases harboring pneumococci, Type II in 1.2 per cent., an Atypical II in 11.2 per cent., Type III, in 13 per cent. and Type IV in 73.8 per cent.

160 (1338)

Renal action in acute nephritis.

By DANA W. ATCHLEY (by invitation).

[*From the Coolidge Fellowship and the Medical Clinic, Presbyterian Hospital, Columbia University, New York.*]

The clinical discussion of these cases will appear with the functional results in a later paper. In the series are six acute cases and two cases entering the hospital in an acute attack but giving a history of previous renal disease. The phenolsulphonphthalein excretion, the renal test day, the blood urea, the urea index and the chloride index were employed for the functional studies. The

[1] Stillman, E. G., *Jour. Exp. Med.*, 1916, XXIV, 651.

renal test day contributed little of value. Nocturnal polyuria was constant but quite independent of the kidneys' ability to concentrate. "Maximal impairment," according to Mosenthal, occurred in two cases, one showing no other abnormalities of renal function, and the other with very moderate impairment, while a fatal case concentrated to 1.022. The balances of salt and nitrogen so frequently included in the performance of this test were not done because of the obvious unreliability of a twenty-four-hour experiment. Moreover, in view of its great dependence upon a factor so variable as the amount of water excreted, systematic determinations of the specific gravity over a brief period must be very carefully interpreted. Many inconsistent phthalein results were found, but in general the value of the test in acute nephritis may be summed up by saying that variations above a level of 20 to 25 per cent. were rarely of a functional significance coinciding with the rest of the picture. A figure below 20 per cent. was of more serious import, although one case excreting only 15 per cent. recovered completely.

The blood urea estimations proved the most consistently valuable means of determining the severity of the disease in any given case. The highest figure was in the only fatal case, the other high figure was undoubtedly the next most severe, while the two cases without any evidence of retention made the most rapid recoveries.

If we overlook variations above the normal and consider the usual rather than the average results, we have in the urea index an expression of the gross renal function that agrees fairly well with the impression gleaned from the other data. But I would hasten to add that even moderately rigid interpretation of the Ambard as a real index of urea function may lead to the greatest error. This criticism is based on such observations as the following. At constant levels of blood urea, an increase in the water excretion in one case caused a 28 per cent. drop in the index (according to McLean's formula), although the rate of excretion (D.) was the same.

Date.	Volume Urine.	Blood Urea.	Rate—D.	Index.
Feb. 28	1,128	0.38	22.5	98
Mar. 14	2,448	0.37	22.4	70

In another case the function was higher when the kidney excreted at a rate of 7 gm. than when it excreted 13 gm., with the blood concentration unchanged for the two determinations.

Date.	Blood Urea.	Urine Conc.	Rate D.	Index.
June 21	0.29	9.4	7.0	32
July 12	0.28	1.7	13.4	27

Other facts could be adduced to demonstrate that Ambard's formulæ are untenable as physiological laws. Whatever clinical value is possessed by the index is due to the fact that a pathological kidney so frequently retains urea and is unable either to concentrate normally or to excrete the usual amount of urea—all factors tending to decrease renal function as expressed by the index.

Numerous estimations of McLean's chloride index were made on these cases but in no case was the rate of excretion clearly dependent upon the concentration of chlorides in the plasma. In general the latter remained fairly constant, as soon as equilibrium was established after diuresis, while the urinary output was proportional to the daily intake. With identical plasma chlorides the rate of excretion showed the widest variations under different dietary regimes. For example, with a constant plasma concentration, one case excreted at a rate of 0.6 gm. on a salt-poor diet and at 13.6 gm. when 4 gm. of salt were added daily to the diet. Other cases showed the same picture, a picture difficult to ascribe to changes in the course of the diseases. The reaction to a single administration of 7 gm. of salt was interesting. There was a rise of 0.2 gm. per liter in the plasma concentration with no change in the rate of excretion; but forty-eight hours later the rate of excretion had increased seven times, although the plasma was unchanged.

I wish to call your attention to a pertinent experiment upon a normal individual. Table I is self-explanatory. There is a marked increase in the threshold with the assumption of a salt-poor diet; in fact, few nephritics show so high a threshold. One of the most striking things is the actual increase in plasma concentration coincident with a decrease in urinary salt output. This experiment I have been unable to repeat (for lack of time), but if confirmed it is sufficient evidence that Ambard's laws of

salt excretion are quite false. Taken with the findings in this series, it quite disproves the assumption that the rate of excretion is dependent upon the height of the plasma concentration above a hypothetical threshold. The evidence here presented seems to indicate that we have quite a constant level in the plasma with a urinary excretion independent of the blood figure and in general dependent upon the amount of salt ingested. This suggestion leaves us still in the dark as to the actual renal mechanism, but opens several interesting leads that I have not time to discuss.

Since any so-called threshold determined during a salt-poor regime is so entirely misleading, even in the normal, the frequency of that regime in the treatment of nephritis makes the clinical application of the index rather limited. Much more valuable than what the formula expresses are the facts that may be obtained from a determination of the plasma chloride concentration together with a knowledge of the daily chloride balance. There is a distinct tendency to find a higher plasma concentration than normal in nephritis and when this elevation occurs I feel that it is significant of an impaired salt function.

As applied to this series, a determination of the blood urea gave the best evidence as to urea function, and total function, while the plasma chloride, together with the daily chloride balance, was the most accurate index of salt function.

TABLE I.

Date.	Diet.	*C.*	*D.*	Actual Plasma NaCl.	Calculated Plasma NaCl.	Threshold.
Apr. 15, 11–1 P. M....	Regular unlimited diet	15.8	10.3	6.10	6.02	5.79
1 P. M....	7 gm. NaCl	—	—	—	—	—
2–4 P. M....	—	17.6	14.8	6.06	6.11	5.57
4–6 P. M....	—	16.6	16.7	6.33	6.13	5.81
Apr. 16.....	Salt-poor diet	—	—	—	—	—
Apr. 19.....	"	5.8	2.5	6.38	5.77	6.22
Apr. 21.....	"	3.2	2.1	6.19	5.74	6.07
Apr. 23.....	"	3.5	1.68	6.00	5.73	5.89

161 (1339)

The application of the Auer-Kleiner morphine test in human diabetes.

By ALBERT A. EPSTEIN.

[*From the Mt. Sinai Hospital, New York City.*]

Auer-Kleiner[1] have found that the administration of morphine of definite dosage to dogs, in which the pancreas had been injured experimentally, caused a very marked rise in the blood sugar, lasting several hours. The administration of opium and its derivatives in the treatment of diabetes mellitus has been a common practice for many years. Many noted observers ascribe beneficial results to this agent. Even today opiates are commonly used in diabetes, particularly in painful conditions, which often complicate this disease.

The observations of Auer and Kleiner are therefore of interest and importance, not only from the standpoint that the administration of morphine to diabetic suspects might (in a very simple manner) give a clue as to the existence of the disease, but they would throw light also on two other phases; namely, (1) the relation of glycosuria to pancreatic disorders; (2) the effect of morphine on the course of the hyperglycemia in diabetes. In other words, if morphine is capable of superinducing a hyperglycemia in animals with pancreatic injury, the increase of a hyperglycemia after the use of morphine in a diabetic individual or its development in a diabetic suspect, would indicate, on the one hand, that a pancreatic disturbance is present; on the other, that the beneficial effects of morphine (as regards pain, etc.) are accompanied by undesirable developments.

I have applied this test in a number of diabetics, both in the sugar-free and the glycosuric states, with the object of determining whether or not the results obtained in experimental animals occur in the human being. The dose of morphine used, given hypodermically, was ¼ of a grain (0.015 g.). This dose is relatively small compared to that used by Auer and Kleiner, in their experiments on dogs; but it is the maximum dose that I felt justified

[1] Auer and Kleiner, PROC. SOC. EXP. BIOL. AND MED., XV, 1, p. 2, 1917.

in administering to patients. The other conditions of the test were like those observed by Auer and Kleiner. The tests were conducted in the following manner: The patients were kept in bed, and after a night of rest and fasting were given a cup of black coffee early in the morning; no other food was given. At noon a specimen of the patient's blood was taken for blood sugar analysis; another at 12.30; a third, at one o'clock; and two others, at hourly intervals. This constituted the control day, as no morphine was given. At the end of this period, the patient received food. On the following day, the tests were made in exactly the same order, with the addition that directly after the removal of the first specimen of blood, a dose of mrophine was given. The accompanying chart shows the maximum and minimum effects produced by the morphine. It is evident that in the case showing the maximum change (Example 1) a certain elevation of the blood sugar occurs. This elevation, however, is very slight, compared to the changes observed by Auer and Kleiner in dogs. The rise here observed would not be sufficient to base the conclusion that a mobilization of sugar occurred in consequence of the morphine administration. In a majority of the cases observed, the change in the hyperglycemia was even less marked than this. The other case presented (Example 2) shows the course of the hyperglycemia before and after the administration of the morphine. In this instance, as in many others, the morphine is without any effect at all.

The results of the observations which I present here do not in any way militate against the results obtained by Auer and Kleiner in their experiments. The discrepancy may be due to a difference in the size of the dose (per kilogram body weight) of morphine used in the tests made by Auer and Kleiner, and those made by me. It is questionable whether the method can have the clinical application which was expected, unless one desires to exceed the rules of therapeutic usage, in regard to the morphine. The effect of the morphine upon all the patients tested was pronouncedly narcotic.

I have intentionally omitted to discuss the question of the effect of morphine on the glycosuria because the results were too variable to admit of any conclusions.

MORPHINE TEST.

Blood Sugar.

Time.	Per Cent.			
	Example I.		Example II.	
	Control Day.	Test Day.	Control Day.	Test Day.
12.00	0.180	0.180	0.156	0.150
12.30	0.182	0.202	0.132	0.135
1.00	0.182	0.216	0.135	0.140
2.00	0.176	0.180	0.135	0.128
3.00	0.180	0.179	0.138	0.123

162 (1340)

A method for determining minute quantities of nitrogen in nitrogenous substances.

By THEODORE KUTTNER (by invitation).

[*From the Department of Physiological Chemistry, Mt. Sinai Hospital.*]

When an alkaline mixture containing phenol and NH_3 or NH_2 groups is oxidized by a hypochlorite, a blue-colored solution results. This color is produced in the cold in mixtures of proper concentration, while mixtures of more dilute solutions give no color or only a very faint blue. Upon heating the mixtures, those containing the minutest trace of the above-mentioned groups develop a color, while the color of the more concentrated ones deepens considerably in intensity. The latter varies in direct proportion to the amount of the NH_3 and amino acid group present.

This reaction is made the basis of colorimetric methods for the estimation of incoagulable nitrogen, urea and ammonia nitrogen in blood, urine and other biological fluids, and for the estimation of certain substances containing amino groups such as albumen, arsphenamin (salvarsan), etc. The estimation is carried out by comparison with a suitable set of standards or in a colorimeter against a known standard.

In estimating incoagulable nitrogen of the blood, the method briefly is as follows: the proteids are precipitated either by the addition of 2.5 per cent. trichloracetic acid to 10 times the volume

of blood taken or by a method described elsewhere,[1] *i. e.*, by boiling the desired quantity of blood after the addition to each c.c. of blood taken, of 5 c.c. of water, 1 drop of 10 per cent. acetic acid and 2 drops of saturated solution of sodium acetate, cooling and adding a few drops of 5 per cent. colloidal iron and water up to 10 c.c. In either case after filtering, 1 c.c. of water-clear filtrate equivalent to 0.1 c.c. of blood is treated in a micro-Kjeldahl. After the reaction is completed, it is neutralized and water is added up to 5 c.c. The color reaction can then be produced directly in the tube after the addition of the reagents as follows:

1. Add 1 c.c. of phenolate soda solution. (40 gm. phenol, 50 c.c. of 40 per cent. NaOH solution, water up to 100 c.c. mixed in the cold.)

2. Add 1 c.c. of 2 per cent. hypochlorite soda solution. Mix well after addition of each reagent and add simultaneously to a known standard or to each of a set of standards containing 5 c.c. of water with 0.01 mgm., 0.02 mgm., 0.03 mgm., etc., of ammonia nitrogen and place simultaneously with the unknown in boiling water for five minutes. They are then allowed to cool, poured into a graduate, made up to 10 c.c. with water and the unknown compared with the known standard or standards.

In case of ammonia the test is sensitive enough to detect and estimate 1 part of ammonia nitrogen in a dilution of about 10,000,000.

Details of the various methods will be published in the *Journal of Biological Chemistry.*

163 (1341)

Asynchronism of the respiratory movements in lobar pneumonia.

By WARREN COLEMAN.

[*From the Department of Medicine, University and Bellevue Hospital Medical College.*]

Asynchronism of the respiratory movements occurs in many severe cases of lobar pneumonia. The phenomenon was first observed by the author some twenty years ago. A careful search

[1] Proc. of A. A. A. S., Dec., 1916, Theodore Kuttner, "A Modification of Folin's Method for the Estimation of Creatinin in Blood."

of the literature has yielded only two references to the phenomenon, one by Grocco in 1904, the other by Grocco's pupil, Frugoni, in 1910.

The phenomenon consists in the separation of the contractions of the diaphragm and intercostal muscles by a definite, though variable, time-interval. As far as has been observed, the movement of the abdomen precedes that of the thorax. In well-developed types of the phenomenon the abdominal wall may have reached the expiratory phase before the intercostal muscles begin to contract. In other instances, the lower intercostal muscles take up the movement of the abdomen and the contraction spreads as a wave over the thorax.

Asynchronism of the respiratory movements has so far been observed only in lobar pneumonia. It possesses grave prognostic significance. Nearly all patients who develop it die. Usually it appears late in the disease. Though generally associated with other indications of severity, it may give the first intimation that the disease is likely to be fatal.

164 (1342)

A skin reaction to pneumotoxin.

Charles Weiss and John A. Kolmer.

[*From the McManes Laboratory of Experimental Pathology, University of Pennsylvania.*]

The work reported is a part of the studies on the properties of pneumotoxin and its probable rôle in the pathology of lobar pneumonia. Previous investigators (Clough, Weil, Steinfield and Kolmer) working with dried, autolyzed or heat-killed pneumococci failed to elicit any constant, specific reaction in cases of lobar pneumonia. The present authors used the endocellular hemolytic toxin of the pneumococcus freshly prepared for each test by dissolving the washed living organisms (Type I) in solutions of sodium choleate. Guinea pigs previously sensitized with sublethal doses of pneumotoxin or with the serum or lung exudate of dogs suffering from experimental lobar pneumonia, reacted to the

intracutaneous injection of 0.1 c.c. of the toxin by a local erythema and hemorrhagic edema in the subcutaneous tissue overlying the muscle. The skin reaction to heat-killed pneumococci was negative in most of these animals and when positive, was of a suppurative type, marked by less edema and more leucocytosis. Control animals gave uniformly negative results.

Among human adult cases of lobar pneumonia the reaction (which was characterized by a local edema and erythema) was elicited as early as the fifth and as late as the thirteenth day of the disease (two days before and six days after the crisis, respectively). In children it was demonstrable about the same time, but was negative immediately or one or two days after the crisis. Patients recovering by lysis reacted as late as the thirty-second day. In general, the test was positive in all active cases, that is, throughout the toxemia. Cases earlier than the fifth day of the disease were not available. Control patients, suffering with bronchopneumonia or with acute or chronic infections not of pneumococcic origin, as well as healthy adults and children did not react.

The reaction is regarded as similar to the tuberculin reaction and is indicative of a state of allergy to pneumotoxin. Sensitization to the toxin presumably takes place with its liberation (by the action of normal body enzymes upon pneumococci normally localized in the lung alveoli) at the time of the prolonged chilling due to exposure. Failure to elicit the reaction during convalescence indicates the establishment of a temporary immunity or the disappearance of excess of toxin. This skin test does not seem to be of value as a method of serological type diagnosis but may aid in differential diagnosis between appendicitis or tuberculosis and pneumonia (especially in children). It is also of interest because of its bearing on the mechanism of the crisis.

SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

Ninety-first meeting.

University and Bellevue Hospital Medical College, April 17, 1918.

President Gies in the chair.

165 (1343)

The urea content of the blood.

By LUDWIG KAST and EMMA L. WARDELL.

[*From the Department of Medicine and the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.*]

Although the retention of urea nitrogen in the blood is now generally regarded as a reliable diagnostic sign of faulty kidney function, there still remains a disturbing degree of confusion as to what should be considered the normal and what a pathological urea content of the blood. Repeated observations of Folin and Denis, and those of our own laboratory, seem to prove conclusively that in normal adults the concentration of urea nitrogen is from 12 to 15 mg. per 100 c.c. of blood. A study of the records of routine blood analyses of hospital patients, on the other hand, shows that many individuals without symptoms of kidney lesion have a urea nitrogen of more than 15 mg. per 100 c.c. of blood. Of a series of 244 cases, extending over a period of 5 months, 206, or 84 per cent., had a urea nitrogen concentration of not more than 20 mg. per 100 c.c. Of these 206 cases, 83, or 40 per cent., showed some indications of kidney lesion, while of the remaining 38 cases in which the urea nitrogen was more than 20 mg., 29, or 76 per cent., showed other renal symptoms. Throughout the series the higher blood urea was closely paralleled by the greater degree of kidney lesion, as indicated by presence of casts or albumin in the urine, high blood pressure, high blood sugar and creatinine

or low phthalein output. From these observations we conclude that 20 mg. per 100 c.c. may be taken as upper normal limit of urea nitrogen in the blood of hospital patients.

166 (1344)

Production of transplantable growth.

By RHODA ERDMANN.

[*From the Osborn Zoölogical Laboratory, Yale University.*]

After analyzing the known numerous theories on the origin of abnormal growth and especially of malignant growth, which have been devised either with or without reference to facts, it seems possible to concentrate the apparently necessary postulates for the successful origin of abnormal growth to the following conditions which cover all important theories on this subject. The presence of a cell group in an indifferent stage of growth, the dissociation of this cell group from its surrounding and its change from the indifferent stage of growth to a stage of proliferation, the inflammation of the connective tissue, neighboring this cell group, and a changed metabolism of the cells, in which this cell penetrates and which it destroys. Supposed these conditions are true to actual facts it must be possible to produce experimentally these factors either in various sequence or all at once and observe results.

I. Embryonic tissue of the chicken heart, after twelve days' incubation, was cultivated in a plasma medium for three days, and three 1 mm. square pieces implanted subcutaneously into a chicken. The implants produced after *ten days* small protuberances. Sections show cyst formation, caused by the isolation of the epidermis above the implants and *inflammatory* proliferation of the connective tissue of the host. Cyst formation, destruction of the feather follicles and accessory apparatus of the feather after previous isolation of the feather-forming complexes are visible. The implanted embryonic heart-muscle tissue which is surrounded by lymphocytes and newly formed connective tissue cells shows no considerable development. Protuberances produced in the same manner disappear after three weeks, as was to be expected.

II. The size of such a growth can be enlarged by a few injections of a *growth-promoting* substance, *i. e.*, tetheline, into the protuberance itself. The growth treated with tetheline is three times as big as its untreated neighbor. Sections show the implant, bordering on the unchanged skin, closely connected with it by newly formed tissue. The embryonic heart-muscle tissue particles are visible after 10 days. Newly formed connective tissue penetrates into the subcutaneous connective tissue of the host. The implant is vascularized by the blood vessels of the chicken. In the skin above the implant, the previously described disarrangement of the epidermal parts and the feather follicles has also taken place. But though these growths often reach the size of a hazelnut, they are finally resorbed.

III. The diet of the host, in which the prepared tissue particle was going to be implanted, was now changed from mixed diet to a vitamine-restricted diet. Chickens were fed with corn gluten meal and the same implantation performed. Breasts of chickens of the same age, but fed on different diet, showed a marked difference in the development of the breast muscles which are nearly atrophied in chickens on vitamine-restricted diet. Protuberances appeared after implantation in the same manner, as in birds fed with mixed food. After twenty-four days half of a protuberance was extracted and sectioned. The destruction processes in the skin are nearly finished, all feather follicles are destroyed, and it could be seen during the extraction that the tissue of the growth had penetrated into the underlying tissue of the host. The remaining half of the protuberance gradually disappeared and in its stead a tumor-like growth appeared. It was partially extracted eight weeks after the original implantation, the chicken dying three days after this operation. Horizontal and transverse sections show the penetration of fibrous connective tissue into the striped muscles, their compression and destruction, together with compressed blood vessels and blood sinuses. Where these processes are finished muscular tissue and fibrous connective tissue of surprising structure appear. The hard tumor shows macroscopically deep pockets in which we see the destroyed blood vessels and muscles; they seem to be filled with brown detritus. Part of this original growth could be successfully grafted on

chickens on vitamine-restricted diet. The fourth generation after thirteen days' growth from February 12 to 25, 1918, showed the character of the original growth, especially the capacity to penetrate into the underlying tissues and to destroy them.

I will not omit to point out that the growth described here did not show bacterial contamination. The possible objection, that this growth which is very similar to those described by Rous,[1] 1910, *a* and *b*, and Rous and Lange,[2] 1913 (compare 1910_a, Fig. 5, 1910_b, Figs. 2 and 3, and 1913, Fig. 7) might be a "spontaneous growth," I will refute immediately by saying that the repetition of this series of experiments on a larger scale was successful. Only in two of twelve chickens, which had received (1) implantation of heart tissue, (2) tetheline injections into the implant, (3) vitamine-restricted diet, growths were produced, the other ten, in which one or two different factors are lacking, showed no protuberance formation.

The produced growth, the genesis of which I have outlined, seems to be a connective tissue growth. It is infiltrable, destructive and transplantable. Further cytological investigation, especially the solution of the question, whether the implanted embryonic tissue cells are really forming by their descendants part of the growth, will be necessary to name this transplantable growth correctly.

A detailed description of this growth and two others, together with their descendants, will appear later.

167 (1345)

Immunization of monkeys against poliomyelitis.

By **H. L. ABRAMSON** (*by invitation*).

[*From the Bureau of Laboratories Dept. of Health, N. Y. C.*]

This work represents efforts made toward developing a practicable method of immunization against acute poliomyelitis. Highly potent monkey polio virus is attenuated by heat in two ways. In one case 5 c.c. of brain and cords in 10 per cent. saline

[1] *Rous, Jour. Exp. Medicine*, 1910, p. 285 and p. 397.

[2] Rous and Lange, *ibidem*, 1913, p. 651.

suspension from monkey poliomyelitis is subjected to 55° C. for one half hour and injected subcutaneously into monkeys on ten successive days. This represents the use of killed virus. In the other method the 10 per cent. emulsion is subjected to graded attenuation as follows: heated to 55° C. for one half hour on first two days; to 45° C. on third day; to 37° C. on fourth day and on the fifth day the material is injected unheated.

On the 20th day after the series of subcutaneous injections are completed, the treated animals are bled from the heart in order to test for the presence of neutralizing substances in the blood. On the following day the treated animals were tested by subjecting them to three to five intracerebral lethal doses of highly potent monkey virus. The sera of the treated animals were placed in contact with equal part of 5 per cent. emulsion of monkey virus at 37° C. for two hours and then in ice box over night. On the day of intracerebral test for treated animals, the serum virus mixtures were inoculated intracerebrally into normal monkeys. The same virus emulsion was used in the test inoculations and serum virus mixtures.

The results with killed virus injection method were not so good as with that of graded attenuation. In the former method only 3 animals were used. Of these two succumbed to test inoculations with the usual incubation and the third presented a delayed incubation of 12 days, but recovered with residual paralyses of both arms and legs. The serum of one of these completely neutralized virus; in another the incubation was prolonged to 18 days but animal recovered and now presents residual paralysis of legs; in the third the incubation was prolonged to 10 days and animal died. The incubation periods of two normal controls were 5 and 6 days respectively.

Of eleven animals subjected to injections by the graded attenuation method, none showed any ill effects from the treatment itself. Five out of eight subjected to the intracerebral test injection of from 3 to 6 lethal doses resisted successfully. Sera of all the animals tested demonstrated the presence of neutralizing substances. Three completely neutralized 5 per cent. emulsion of highly potent virus in proportion of 1 : 1. Four others delayed the four- and five-day incubation periods of normal controls to

from 10 to 25 days. To indicate potency of test virus two animals that had recovered from previous experimental infection and which presented residual paralyses succumbed as readily as the normal controls to the intracerebral injection of only three times the dose used to test the treated animals. Animals that have recovered from experimental infections have been found to be highly resistant to reinfection.

The latter method requires only a short time; the material is easily produced and gives a considerable degree of immunity. The test method is far more severe than that to which persons are subjected in the natural infection inasmuch as in this artificial test the defensive forces in the blood stream are circumvented, whereas in the natural infection the infective material would first come in contact with the neutralizing substances present in the blood stream before it could gain entrance to the central nervous system. The natural infection is far less severe than the experimental disease in which we may have a mortality of about 99 per cent.

168 (1346)

Persistence of immunity following toxin-antitoxin injections.

By WILLIAM H. PARK.

[*From the Bureau of Laboratories Dept. of Health, N. Y. C.*]

During the past years, we have immunized a series of guinea pigs and a series of horses and a series of children with diphtheria toxin-antitoxin mixtures. The duration of the immunity presents certain points of great interest. So far as our observation goes, guinea pigs are never naturally immune; horses in the very great majority of cases are immune; adults and infants under 6 months are immune to 80 per cent., while very young children are non-immune to about 60 per cent. We find, in the guinea pig, that active immunity lasts for about nine months. In horses, we find the increased immunity, due to the injections, lasts from nine months to twelve months, at which time the horses return to their original amounts of natural antitoxic immunity.

In testing human beings from month to month, it was a matter of great interest to find whether the infants and children who be-

come immune, due to the injections, would, like the guinea pigs, lose their immunity in about nine months or whether the active immunity would be replaced by natural immunity just as in the majority of infants the stage of lack of immunity is followed by antitoxic immunity. We have found, in infants, two years after successful immunization that the great majority have remained immune, not over 6 per cent. losing their immunity. We have a right, therefore, to hope that the stimulated immunity has been replaced in the very great majority by a natural immunity and that this will hold for life. The facts that the protection lasts and that the injections are harmless make the active immunization of infants appear to be a practical measure of eliminating diphtheria.

169 (1347)

The composition of dried vegetables with special reference to their nitrogen and calcium content.

By MAURICE H. GIVENS.[1]

[*From The Sheffield Laboratory of Physiological Chemistry, Yale University, New Haven.*]

Some of the common vegetables have been dried on steam-heated radiators at 65° C. to 70° C. The loss of water by this method of drying has been found to correspond to the water content of these materials as determined by Atwater and Bryant. Detailed analyses of these will soon be published.

Such dried green vegetables as swiss chard, beet tops, and celery have a high content of inorganic constituents, particularly calcium. Their content in mineral components decidedly preponderates over that of such plant products as carrots, cabbage, sweet potatoes, and potatoes. Attention is called to the possible loss of mineral constituents in preparing vegetables for drying when they are first cooked in water which is discarded.

[1] Seessel Research Fellow in Physiological Chemistry, Yale University.

170 (1348)

Observations on the production of experimental scurvy in the guinea pig.

By BARNETT COHEN (*by invitation*).

[*From the Public Health Laboratory, Medical School, Yale University, New Haven, Conn.*]

The customary method of producing experimental scurvy in the guinea pig with a monotonous cereal diet is open to several points of criticism. The protein may be inadequate, inorganic salts deficient and the physical texture unsatisfactory. Our work led to a search for some good food material capable of dietary analysis; and through the results obtained by Osborne and Mendel[1] and others the soy bean seemed most suitable. Its protein is adequate for maintenance and growth (of the rat) and the only inorganic constituents deficient are Na, Ca and Cl. We used soy bean flour autoclaved at 15 pounds for 30 minutes. To it was added Ca lactate and NaCl. Dried brewer's yeast and fresh raw Jersey milk supplied the two recognized dietary accessories (vitamines). Young guinea pigs on this diet gain in weight faster than on the usual cabbage-carrots and oats ration. After 10 days, while still gaining in weight they develop swelling of the joints and general tenderness—symptoms considered indicative of experimental scurvy. Later occurs loss in weight followed by death. Stools were frequent and pasty; toward the end diarrheal. A marked polyuria was present from the beginning of the diet. Addition of over 7 per cent. cellulose as roughage did not change the result. Orange juice, 5 c.c. daily, or fresh cabbage prevented and cured these symptoms promptly with increase in weight.

171 (1349)

Further experiments with thyroidectomy in Amphibia.

By E. R. HOSKINS and M. M. HOSKINS.

[*From the Department of Anatomy, University and Bellevue Hospital Medical College.*]

The thyroid gland anlage was removed from a large number of *Rana sylvatica* larvæ 6 to 8 mm. in length. From the time

[1] Osborne, T. B., and Mendel, L. B., 1917, *Jour. Biol. Chem.*, *32*, 369.

the larvæ averaged 18 mm. in length (225 experimental and 182 control animals) the experimental larvæ grew more rapidly than the controls, and most of them became ultimately much larger than the controls, the largest being 72 mm. in length as against 50 mm. for the controls. Thirty thyroidless animals failed to metamorphose and in twelve others the thyroid regenerated, permitting metamorphosis at the normal time. The rest of the experimental larvæ were killed or died before this time. Six of the thyroidless larvæ are still alive, nine months after the normal time of metamorphosis. They practically ceased growing six months ago.

In the absence of the thyroid the secretory (anterior) portion of the hypophysis underwent physiological hypertrophy to compensate for the loss of the thyroid, as both normally are concerned with metabolism, although not in an exactly similar way. This hypertrophy probably accounts for the gigantism and infantilism (retention of the larval form) of the thyroidless larvæ. Normally, synapsis begins in the testis after metamorphosis, and in the ovary a short time before metamorphosis. In our experimental animals so far examined, synapsis in the testis was practically completed at the time of metamorphosis of the controls and differentiation of the ovary also was hastened greatly. The hypertrophy of the hypophysis probably accounts for this sexual precocity. The growth in size of the gonads continues with that of the other parts of the body and is independent of the changes normally occurring in metamorphosis. Both processes, however, depend upon the general metabolism. Failure of metamorphosis is due to abnormal metabolism, brought about, either by loss of normal thyroid secreton or by abnormal secretion of the hypophysis which is affected by removal of the thyroid. Both causes may be involved, and also, the secretion of still other glands may be changed after loss of thyroid function. Failure of metamorphosis may depend directly upon an abnormal calcium metabolism with which the thyroid is believed to be concerned, since the initiation of metamorphosis is probably brought about by growth of the skeleton. The condition in thyroidless larvæ may be neotony, as Allen has called it. The hypophysis of axolotls should be investigated to see if it is the cause of failure of metamorphosis in these animals. In the obser-

vations so far made, the hypophysis of the female is much larger than that of the male, both in our thyroidless and control animals. If this point is established by further study, it may account for the fact that the ovary differentiates earlier than the testis.

In one of our experimental animals, the thyroid regenerated, and then hypertrophied. The animal underwent metamorphosis a month before the normal time, and at a size about one third normal. Its ovaries were in a stage of differentiation, much further advanced than normal. Its hypophysis could not be studied on account of poor fixation.

The various other organs and ductless glands of the thyroidless animals are being studied at this time. In general they are all larger than normal, but we have not yet determined whether there is present true hypertrophy or whether the size is merely proportional to the size of the larvæ which are larger than normal.

172 (1350)

Generalized analgesia in cats after exposure to a war gas $(CH_3)_2SO_4$.

By JOHN AUER.

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute.*]

Procedure.—Cats were subjected to the action of dimethyl sulphate fumes in a concentration of 4.5 to 10,000 of air for one hour. The dimethyl sulphate, which is a liquid at ordinary temperatures with a specific gravity of 1.32 and a boiling point of 188° C., was vaporized by an electric hot plate in the gas chamber. An electric fan in the gas chamber kept the available fumes mixed with the air. The temperature of the gas chamber was maintained between 25–30° C.

With this strength of gas the cats usually succumbed in 30–60 hours.

In cats which are gassed as described above a strong generalized analgesia is observed within a few hours after exposure to the dimethyl sulphate fumes, and reaches a high degree within twenty-four hours. If the animal survives, the analgesia may still be well marked six months afterwards.

In such animals severe operations may be performed without any anæsthetic and without eliciting any or only slight indications of pain. Thus, for example, the femoral artery may be dissected free, tracheotomy performed, the vagosympathetic trunk exposed in the neck, the tibialis anticus muscle dissected free, the peritoneum opened and the parietal walls rubbed by an inserted finger, or gentle traction exerted upon the gut, without causing any or at most only slight signs of pain. Severe compression of the tail, toe pads, nasal septum, lip, ears, cause no sign of pain, though the tail is twitched aside, the foot withdrawn, the head turned away or the ears flattened against the head when the pressure stimulus is stopped.

The loss of pain perception was, however, not complete in any animal: blunt dissection of a mixed nerve which stretched it or electrical stimulation of the sensory nerve, always caused more or less restlessness of the animal, but never to the degree which is obtained by the same manipulation in a partially anæsthetized cat.

There is therefore not only an outspoken depression of the peripheral apparatus for pain perception, but the higher centers show also a definite though less marked involvement.

It must be emphasized that the analgesia described above does not depend upon a low general state. There may be a profound general analgesia while the animal is in an excellent general condition; thus in an instance with most pronounced analgesia the blood-pressure was 118 mm., pulse 150, respiration 35 per minute; the tongue and mucous membranes showed a good red color, and the rectal temperature was 37.3° C.

While investigating this analgesic property of dimethyl sulphate gas, Naiding published observations in the *Russky Vrach* (May 6, 13, 20, 1917, XVI) recording the production of local or generalized skin analgesias in soldiers gassed with chlorine and phosgene. Naiding appears to have tested skin sensibility only and does not mention that severe cutting operations can be carried out without pain. In experiments which are at present being carried out with another exceedingly toxic war gas the same remarkable analgesia is produced. Analgesia may thus be produced by at least three widely different war gases and may perhaps be a property of most gases usable for military purposes.

This property, common at least to some war gases, could be utilized practically in gassed soldiers who are also wounded and require surgical intervention. It is very likely that they would need little if any anæsthetic. The gassed soldiers would thus be spared another danger and a valuable drug would not be needlessly expended.

173 (1351)

Localized pulmonary edema in cats after the inhalation of a war gas $(CH_3)_2SO_4$.

By JOHN AUER.

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute.*]

In a number of cats which died or were killed 27–50 hours after gassing with $(CH_3)_2SO_4$, as described in the previous note, a localized pulmonary edema was observed on autopsy. The edema rarely involved the whole lung, but only a certain lobe or portions of a lobe showed a striking degree of pulmonary edema. The lobe involved was most frequently an upper or a middle lobe, but the lower lobes were by no means exempt. The best example of edema of a portion of a lobe was noted in the right middle lobe, where a section near the hilus showed extreme pulmonary edema, while the rest of the lobe was only moderately edematous. It was by no means infrequent to find one lobe fairly saturated with fluid while the rest of the entire lung tissue was practically free from fluid. Hemorrhages and pulmonary congestion, it should be noted, are not prominent features of the autopsy picture of the lungs after gassing with $(CH_3)_2SO_4$.

The causation of this localized pulmonary edema obviously must be due to some mechanism which affects the lung chiefly at the site involved; it cannot involve the whole lung to the same degree, for then the whole lung would have to be equally edematous. This locally acting mechanism is apparently the combination of a partial or complete stenosis of a bronchus or bronchiole with inspiratory dyspnea. These conditions are realized in cats gassed with dimethyl sulphate, for a marked inflammation of the respiratory passages with pseudo-diphtheritic membrane formation

occurs, and inspiratory dyspnea is always present to a greater or less degree.

Under these conditions therefore each alveolus which is in connection with a stenosed bronchus or bronchiole will act like a miniature dry cup during inspiration, because the pressure in these alveoli will decrease as the intrathoracic pressure decreases during each inspiration, for little or no air enters through the stenosed air passage. Therefore during each inspiration, which is slow, labored and prolonged in the gassed cat, the capillaries of the alveolar walls are subjected to an aspirating action which facilitates or initiates the passage of a transudate into the alveolar spaces. The production of this transudate is probably also aided by a local damage of the alveolar epithelium which the war gas produces.

It is thus seen that the fundamental conditions which apparently cause the local pulmonary edema of dimethyl sulphate are the same which are also operative in adrenalin pulmonary edema, as Gates and I have described elsewhere.[1]

Practically these observations may be of some value. In gassed soldiers all inspiratory dyspnea should be ameliorated as much as possible, by tracheotomy and artificial respiration if necessary. At another time I hope to report concerning the marked beneficial action of tracheotomy in fulminant cases of pulmonary edema produced by a different type of war gas.

174 (1352)

On the electrical resistance and permeability of tumor tissues.

(Preliminary communication.)

By G. H. A. CLOWES.

[*From the Gratwick Laboratory, Buffalo, N. Y.*]

Preliminary determinations of the electrical conductivity of primary breast carcinomas in mice (supplied by Mr. Millard C. Marsh) indicate that cancer tissues are more permeable to ions than are normal tissues and that the permeability bears a definite relation to the speed of growth, rapidly growing tumors exhibiting

[1] *Jour. Exp. Med.*, 1917, XXVI, p. 215.

a lower resistance than slowly growing tumors. Similar determinations on plant galls (supplied by Dr. Irvin F. Smith) indicate that plant tumor tissues are uniformly more permeable than normal plant tissues, the tumor tissues frequently exhibiting, under reasonably comparable conditions, a conductivity more than twice that of normal tissues. The observations of McClendon, Gray, etc., that sea urchins' eggs exhibit an increased conductivity during the first stages of development harmonize with the above observations on cancer tissue and lend support to the conclusion previously reached by the writer, as a result of chemical analysis of mouse tumors and blood reactions exhibited in cancer and pregnancy, that both normal and pathological proliferative processes depend upon an increased permeability of the protoplasmic film to water and water-borne food stuffs.

An attempt will be made in the next paper to indicate briefly by means of a physical model, the mechanism whereby changes in protoplasmic permeability may be induced by changes in chemical and physical environment and in a subsequent paper to apply the principles involved to cancer.

175 (1353)

On the action exerted by antagonistic electrolytes on the electrical resistance and permeability of emulsion membranes.

(Preliminary note.)

By G. H. A. CLOWES.

[*From the Gratwick Laboratory, Buffalo, N. Y.*]

Artificial emulsion membranes suitable for electrical conductivity and permeability experiments may be prepared by interposing layers of filter paper saturated with an emulsion of oil in soap between supporting sections of rubber tubing in a glass U-tube of the type commonly employed for electrical conductivity determinations. A thicker layer or film of emulsion is generally preferable and may be prepared by introducing into the U-tube a section of rubber tubing of any desired length which is then filled with emulsion. Retaining layers of filter paper above and below

are held in position by additional supporting sections of tightly fitting rubber tubing. Emulsion membranes of this type when exposed to the influence of various antagonistic electrolytes exhibit variations in electrical conductivity and permeability corresponding almost exactly with those observed by Osterhout in the case of laminaria under similar working conditions. For example, .52 M NaCl causes a rapid rise in the conductivity of a saturated filter-paper membrane until the level of the environing solution is almost reached, while .278 M $CaCl_2$ causes first a considerable fall in conductivity which is followed subsequently by a rise to approximately the same level as in the case of NaCl. The most remarkable paralleling of Osterhout's results may be obtained by exposing emulsion layers or films to brief alternating treatments with NaCl and $CaCl_2$. As in the case of laminaria, alternating variations in conductivity within comparatively wide limits may be obtained without any apparent injury to the membrane which may subsequently be returned to sea-water or a balanced solution of NaCl and $CaCl_2$ and exposed to a similar treatment the next day. However, just as in the case of laminaria, too prolonged an exposure to either NaCl or $CaCl_2$ may cause changes in electrical conductivity and permeability beyond the critical point at which recovery is still possible and the membranes subsequently exhibit extremely erratic results or fail entirely to respond to treatment. That electrical conductivity experiments on membranes of this type or living tissues afford an index of their permeability to water and water-borne substances was demonstrated by paralleling the above experiments in the following manner: Layers of emulsion supported between filter paper and rubber tubing were introduced into a series of long glass tubes and the speed with which distilled water, sea-water and solutions of NaCl and $CaCl_2$ and a balanced mixture of NaCl and $CaCl_2$ flowed through the membrane was determined by measuring the fall of the fluid in each tube at given time intervals. The distilled water, sea-water and suitably balanced mixtures of NaCl and $CaCl_2$ flowed through the membranes at approximately equal speed while NaCl flowed through the membrane at a vastly greater speed and $CaCl_2$ at a somewhat reduced speed. The relative speeds of flow of various solutions were found to correspond

to an extraordinary degree with data previously accumulated by the writer by means of the capillary pipette drop method regarding the influence exerted by NaCl, $CaCl_2$, etc., on soap films and surface tension. These conductivity and filtration experiments taken in conjunction with the recently published filtration experiments of Hirschfelder and the writer's previous experiments with soap films and emulsions lend strong support to the contention previously advanced by the writer that variations in the permeability of the protoplasmic membrane are attributable to the action of electrolytes and metabolic products on delicately balanced interfacial soap films and emulsion systems and that proteins may play no part in the valve-like mechanism controlling permeability other than to afford a supporting filamentous or mesh-like structure.

Further support of this point of view is found in the fact that when blood plasma is clotted by the addition of $CaCl_2$ no considerable change in electrical resistance is noted while in the transformation of an emulsion of oil in water into one of water in oil by shaking with $CaCl_2$ the resistance suddenly rises to an enormous extent at the critical point at which oil becomes the continuous or external phase.

It is obvious, therefore, that while the clot or jelly formed from fibrinogen is almost as freely permeable to water as the original plasma, the emulsion under similar conditions has been converted from a system which is freely permeable to one which is absolutely impermeable to water. Any intermediate degree of permeability is theoretically obtainable, providing the working conditions are sufficiently delicate. Since the main factor involved is that of surface tension, a finely dispersed emulsion structure contained in the capillary spaces of a jelly or its filter paper analogue would obviously respond with far greater delicacy to those substances which promote or inhibit permeability than would an emulsion in gross form. Since the whole question resolves itself, apparently, into one of the state of dispersion of soaps it is obvious that the dispersing effects exerted by NaCl on soap films and the consequent considerable lowering of the surface tension of the water phase and promotion of the permeability of the emulsion which have already been demonstrated in previous communications by means of the capillary pipette and other methods would be even more

pronounced in capillary spaces. Conversely calcium chloride, which causes an aggregating effect on soap films raises surface tension and tends to diminish the permeability of emulsion systems to water, would tend to diminish the permeability of the structure as a whole to an even greater extent when functioning in capillary spaces.

The above data correlates admirably with the well-known fact that alkalis, salts of sodium, potassium, etc., promote the permeability of tissues while salts of calcium, magnesium, and other di- and trivalent cations exert the reverse effect. Also with the observations of Beebe and the writer regarding the high K content and low Ca content of rapidly growing tumors and the low content of K and high Ca content of slow-growing or stationary tumors.

The experimental data on which this paper is based together with a full development of the theoretical aspect of the case from the standpoint of surface tension and tissue permeability will shortly be published.

176 (1354)

The relationship of the leucocyte count and bone-marrow changes in acute lobar pneumonia.

By ROBERT A. LAMBERT and S. S. SAMUELS.

[*From the Pathological Laboratory of the Presbyterian Hospital, New York City.*]

It is well recognized that the leucocyte count in lobar pneumonia may vary within wide limits. The majority of the cases which end fatally show either a very high or relatively low count, while those with favorable outcome most often have counts between these extremes.

For the low leucocyte count, at least two explanations have been suggested: (1) the bone marrow fails to react, either as the result of some previous injury (chronic alcoholism, for example), or on account of a paralysis of the blood-forming elements from overstimulation by the pneumococcus infection itself; (2) a rapid spread of the pneumonic process takes the leucocytes out of the blood faster than they are thrown into the circulation from the bone-marrow, therefore, the number of circulating leucocytes may

be normal or slightly increased, even though the output of the bone-marrow factory may be far above normal. Still another possibility must be conceived, namely, that leucocytes may be formed in some other organ than the bone marrow, for example, the spleen. Dr. F. A. Evans, working in our laboratory, pointed out several years ago that the gray, acute splenic tumor of lobar pneumonia contained large numbers of cells of the myeloid series, as indicated by the oxydase reaction, and suggested that the spleen might be the source of certain of the cells of the pneumonic exudate.

In the present study, an attempt has been made to correlate the bone-marrow changes and the blood picture in twenty fatal cases of lobar pneumonia, which have come to autopsy. In nearly all of the cases, several leucocyte counts were made. In the majority, there was a record of a count on the last day of the patient's life. Specimens of bone marrow were taken from the upper third of the femur in all cases, and paraffin sections studied after hematoxylin-eosin and Giemsa stain. The result of the study may be summarized as follows:

A close parallelism in the leucocyte count and the degree of hyperplasia of the marrow was found in only about half of the cases. A few showed a relatively inactive or aplastic marrow, with a leucocyte count well above normal. On the other hand, there were several cases in which the leucocyte count was consistently low during life, but in which a markedly hyperplastic marrow was found at autopsy.

To explain aplastic marrow in the femur (a condition probably common to all the long bones in these cases) associated with a leucocytosis, we may assume either a hyperplasia of the marrow of the flat bones only, or a formation of leucocytes outside the marrow, most probably in the spleen. The second explanation we consider the more plausible. The cases of marked hyperplasia of the marrow with low leucocyte count are not easily interpreted. We could not find evidence in these cases of such rapid spread of the lesion as would account for a low leucocyte count through an excessive draining of these cells out of the circulation.

ABSTRACTS OF THE COMMUNICATIONS, PACIFIC COAST BRANCH.

Twentieth Meeting.

San Francisco, California, April 8, 1918.

177 (1355)

Anatomical changes produced by repeated intravenous injections of streptococci in rabbits.

By **W. OPHÜLS** and **ELMER W. SMITH.**

[*From the Leland Stanford University.*]

Fifty rabbits received repeated intravenous injections of streptococci with the object in view of ascertaining what subacute and chronic lesions might be produced in this way, the idea being to simulate as much as possible chronic streptococcic septicæmia.

The streptococci were obtained from about two dozen different sources in the course of routine bacteriological examinations at autopsies, and surgical material. The doses injected were moderate. The injections were repeated as frequently as the animals seemed to be able to tolerate them. The animals lived from a few days to 261 days. The results were as follows:

Fourteen animals developed subacute septic endocarditis. The lesions were most commonly found on the left side of the heart, but the tricuspid valve was involved three times and there was severe infection of the pulmonary valve in one. All of the streptococci which produced endocarditis were of the non-hemolytic type. There was only one case in which arthritis and endocarditis occurred in the same animal.

Lesions of the myo-cardium were observed fourteen times. In four cases there were abscesses, in two purulent myocarditis, in two necroses, and in six chronic myocarditis.

There were thirty-one joints affected in ten rabbits. These joint lesions were often very severe and extensive, causing considerable destruction in the cartilage and synovial membrane and in a considerable number of cases involved the neighboring tendon sheaths to such an extent that some of the tendons were entirely necrotic. These joint lesions were produced by both hemolytic and non-hemolytic streptococci.

The musculature was remarkably free from all chronic changes, which is all the more surprising in view of the frequency with which acute changes have been described after intravenous injection of massive doses of streptococci.

In seven of the animals there were thromboses in the blood vessels of the lung, of which six were distinctly septic. Most of these thromboses followed the injection of non-hemolytic streptococci and they were most frequent after injection of the more virulent types of these organisms. Besides these lesions which were evidently produced by the injections, a considerable number of the animals died of severe bronchial pneumonia.

Seven rabbits developed renal lesions showing diffuse acute and subacute glomerulitis, frequently associated with more or less degeneration of the epithelium. No severe interstitial changes were observed. Three of these animals had endocarditis at the same time and three others amyloid degeneration.

The liver of a great many of the animals was diseased, but on account of the frequency with which rabbits suffer from coccidiosis it was impossible to determine whether any of the chronic changes were due to the injections. Some of the animals showed septic thrombi in the blood vessels and recent necroses of the parenchyma which were evidently caused by them.

The gall-bladder was infected in a great many rabbits and some of these cases showed as a result acute cholecystitis; others more chronic changes. Gall-stones were not found in any of the cases.

Fourteen cases showed pigmentation of the spleen or other evidences of blood destruction. Ten rabbits showed amyloid accumulations in the malpighian bodies. The earliest case of amyloid occurred on the forty-fourth day.

It is of some interest to note that four rabbits at autopsy showed an acute tonsillitis which however was probably due to secondary infection.

One of the rabbits developed an acute appendicitis and another an acute ulcer of the stomach. One rabbit died of intussusception.

Endarteritis was observed in four cases; in two of these the lesions were in the branches of the pulmonary artery; in one in the aorta and in one in the branches of the coronary artery. The lesions were comparatively recent and not very severe.

Examination of the central nervous system showed only slight lesions in the pia in a few places. One of the rabbits died as the result of an acute subdural hemorrhage.

So far as the organs of special sense are concerned, a few rabbits developed conjunctivitis, rhinitis and one otitis media. These were all probably due to secondary infections.

One rabbit developed a septic necrosis of the testicle; another one accidentally received a fracture of the rib which was apparently infected by way of the blood stream.

In conclusion it may be stated that the streptococci did not seem to exhibit any special affinity for the organ from which they were isolated nor did any of the strains show any particular affinity for special organs in the rabbits injected except for the fact that the non-hemolytic group seemed to favor the heart valves.

The details of these experiments will be published elsewhere by Dr. E. W. Smith.

178 (1356)

On serum proteins.[1]

By LILLIAN J. ELLEFSON and CARL L. A. SCHMIDT.

[*From the Hearst Laboratory of Pathology and Bacteriology and the Department of Biochemistry of the University of California.*]

Intrapleural injections of aleuronat suspensions into rabbits caused a marked rise of serum globulins and a consequent upset of the protein quotient. Injection of ten cubic centimeters of blood serum from these animals into normal rabbits did not produce appreciable changes in the serum proteins of the latter animals. Apparently there is no factor concerned in the upset of the normal protein quotient which can be transmitted by injection of such homologous sera.

Injection of ten cubic centimeters of 1 : 30,000 homologous typhoid agglutinating serum intravenously into a normal rabbit caused a very slight, non-persistant rise of globulins in the serum of the injected animal. A normal rabbit which received ten cubic centimeters of 1 : 7,000 homologous staphylococcus agglutinating

[1] Aided in part by a grant from the George Williams Hooper Foundation for Medical Research.

serum showed no change in the protein quotient. The results are essentially in accord with previous work on the non-dependence of immunity on the ratio of serum proteins. Apparently, to produce a decided change in the protein quotient a substance must be injected which causes a marked reaction in the injected animal. This is not accomplished by injection of homologous sera.

SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

Ninety-second meeting.

Schermerhorn Hall, Columbia University, May 15, 1918.

President Gies in the chair.

179 (1357)

The dynamics of cell-division.

By **H. H. LAUGHLIN** (by invitation).

[*From the Eugenics Record Office.*]

From the structural point of view mitosis is a process by which a living cell, through continuous and orderly transformations, divides so that (*a*) in equational division each daughter cell duplicates the mother cell exactly at the latter's comparable stage of existence; or (*b*) in ontogenetic division each daughter cell duplicates the mother cell at the latter's comparable stage of existence, exactly in chromatin content, but not necessarily exactly in any other detail.

From the dynamical point of view mitosis is a process related and characterized as follows: Beginning in a single living cell at a stage of high metabolic activity but of divisional stability thence continuing by means of metabolism to a condition of low metabolic activity and divisional instability the mitotic potential is established. Thence reactions still proceed by virtue of the cell's self-contained structural and chemical organization, and its environmental complex. These successive reactions are so timed, localized, and successively interdependent upon their preceding and adjacent activities, that in equational division mitotic stability and metabolic activity are again achieved only when this self-contained

process-chain of reactions reaches a stage wherein are found, in place of one cell, two cells, which if the division be equational are exact duplicates of the one parent cell at the same stage. If, however, the mitosis be ontogenetic the daughter cells will exactly resemble the parent cell at the latter's comparable stage in chromatin content only; their cytoplasmic form and functions may vary greatly.

The structural stages and ends of the mitotic process have been analyzed much more thoroughly than have the dynamical. The analysis of the latter seeks not to find form but to explain what is happening in terms of interplay of forces. It should yield even greater returns in understanding vital and evolutionary phenomena. In attempting such an analysis it is necessary first to learn how to measure[1] a characteristic of mitotic activity, such as its velocity in passing through successive stages. The actively dividing cell must then be subjected to definite measures and quality of light, heat, gravity, pressure, electricity, or chemical influence, and next the behavior of the dividing cell as modified by the definite quantitative and qualitative influences must be measured. We may then examine the behavior of such elementary processes as have been measured while reacting to these definite influences. It is finally in order to seek for similarity of behavior which, when found, suggests very strongly similarity of organization and forces. The completeness and accuracy of such an analysis depends, of course, upon the degree of refinement in the isolation and measurement of factors and the exactness of the parallel to known processes.

This takes us immediately into the realm of physical chemistry. The present paper will discuss a definite problem in this study, namely, the influence of temperature upon the dividing root-tip cells of the onion, and will then seek a preliminary or working analysis by comparing the reaction thus found with similar reactions which physical chemists have found in other and simpler complexes. The following table shows the effect of temperature upon the velocity of the several mitotic stages calculated in the usual Q_{10} form of the chemist.

[1] Laughlin, H. H., "The Duration of Mitotic Stages in the Root-tip Cells of the onion," Carnegie Inst. of Wash. Pub., No. 267.

Mitotic Stage.	Q_{10} Velocity at 20° C. Compared with Velocity at 10° C.	Q_{10} Velocity at 30° C. Compared with Velocity at 20° C.
1. Early prophase	−1.1340 (.8818)	+1.1525
2. Early prophase	+2.6832	+4.9406
3. Mid-prophase or spireme	+2.9599	+2.6404
4. Late prophase	+1.3859	+2.7593
5. Metaphase	+1.4071	+3.0663
6. Early prophase	−1.1701 (.8546)	+2.3440
7. Mid-anaphase	+1.1523	+2.7571
8. Late anaphase	+1.6334	+2.6038
9. Early telophase or di-spireme	+1.3329	+2.1694
10. Late telophase	+1.1240	+3.0931
Resting or metabolic	+1.2215	+4.9463
Stages 2 to 10 inc.	+2.0476	+3.2311
Stages 1 to 10 inc.	+1.1990	+1.3962
Entire cycle, *i. e.*, the 1 resting and the 10 active stages	+1.2139	+2.6218

Next we shall list and classify according to the sign of their temperature coefficients such other elementary and complex physical, chemical and physiological processes as have been described in the literature of the subject, and which may throw light upon the nature of the temperature-reactions shown in the above table.

A. The following forces and processes within medium ranges of temperature (5° C. to 35° C.) react in the positive direction to temperature-increments, that is, they have the Q_{10} value with the + sign:

1. Velocity of chemical reaction.
   In homogeneous reactions $Q_{10} = +2.0$ to $+3.0$.
2. Osmotic pressure. (Always low in colloids.)
3. Plasmolysis. (Protoplasmic permeability.)
4. Diffusion of fluids. (Always low in colloids.)
5. Electric conductivity of solutions.
6. Migration rate of ions.
7. Ionization.
8. Rate of conduction of nerve impulse.
9. Rapidity or rhythm of electric phenomenon.
10. Velocity of protoplasmic streaming.
11. Coagulation.

[1] Loc. cit.

12. Speed of destruction of spores of anthrax by carbolic acid.
13. Velocity of ontogeny in the frog.
14. Velocity of bulk increase in maize seedlings.
15. Rate of digestion in cold blooded vertebrates.
16. Rate of respiration in *Pisum sativa*.
17. Activation of star-fish eggs by chemical agents.
18. Velocity of CO_2 assimilation by the leaves of the cherry-laurel.
19. Expansion of liquids.
20. Vapor pressure.

B. The following forces and processes show the negative coefficient to temperature-increments. Q_{10} values with the $-$ sign.

1. Surface tension.
2. Polarization capacity.
3. Electromotive force of electro-chemical cell.
4. Electromotive force of nerve.
5. Duration of latent period of smooth muscle.
6. Solubility of gases in water.
7. Duration of life in *Drosophila*.
8. Elasticity.
9. Viscosity (absolute internal friction) of dog serum.
10. Cohesive properties generally.

C. Besides these two classes there is another group of activities which shows a positive or a negative coefficient, depending upon conditions, $Q_{10} = \pm$, as follows:

1. Osmosis.
2. Dialysis.
3. Cataphoresis.
4. Tendency toward precipitation.
5. Solubility of salts:

   Most salts $+$
   $Na\ SO_4$ $-$
   Many organic salts $-$

6. Strength of linkage in the second chromosome of *Drosophila*, $-$ at low temperatures, $+$ at high temperatures.

Of the processes here listed[1] 8 were secured from Kanitz's compilation,[2] 8 from Snyder's,[3] one—that of Q_{10} values of the several mitotic stages—was supplied by the experiments of the writer,[4] and the remainder were found either in isolated cases in the literature of related subjects, or one current in text-books.

These are the materials at present available for beginning the analysis of the dynamics of mitosis by means of differential temperature-reactions. The question is, which, if any, of the more elementary of these forces, and what other forces, in what combination and to what degree, are active in each stage of mitosis? Certainly for the most part the several mitotic stages are manifestations of chemical and physico-chemical forces showing a positive index.

Stages Nos. 1 and 6 deserve special attention. Stage No. 1 is the earliest prophase; Stage No. 6 is that in which the chromosomes have left the equator but have not yet reached the poles. Both of these stages manifest a negative coefficient from 10° C. to 20° C. The activities of these stages within such temperatures must, therefore, as shown by the behavior of their resultants of forces, be composed of those processes most of which, or at least the most effective of which, react negatively to temperature increments. This fact alone halves the complexity of the puzzle for these particular mitotic stages, and sets them apart as being characterized by distinctive sets of forces.

Another general observation is worthy of note, physiologists,[5] point out that in simple chemical processes Q_{10} values are greater in the upper ranges of temperature than in the lower, while in vital processes generally the reverse is true. In mitosis the complex of forces is such that in most stages a given increment of temperature will cause a greater velocity-response in the particular

[1] The references to each of the experiments providing the listed data on Q_{10} values will be given in the completer paper now being prepared by the writer.

[2] Kanitz, A., "Temperatur und Lebensvorgange." Berlin, 1915.

[3] Snyder, Charles D., "A Comparative Study of Temperature coefficients of the Velocities of Various Physiological Actions," *Am. Jr. Physiol.*, Vol. XXII, Aug., 1908.

[4] Loc. cit.

[5] Harvey, E. Newton, "Effect of Different Temperatures on the *Medusa cassiopea*, With Special Reference to the Rate of Conduction of the Nerve Impulse." Carnegie Inst. of Wash. Pub. 132. 1910.

ranges of vital temperatures than in the lower. This indicates by the method of the chemist that which the cytologist has long held, namely, that the mitotic complex is something more than a sum total of continuous and independent physiological actions; it is an interrelated system of forces, vastly complex and which system varies in its complex at each stage of mitotic progress.

180 (1358)

Diet and roughage in relation to the experimental scurvy of guinea pigs.

By BARNETT COHEN and LAFAYETTE B. MENDEL.

[*From the Sheffield Laboratory of Physiological Chemistry, Yale University, New Haven, Conn.*]

It has been repeatedly demonstrated that exclusive diets of cereals produce scurvy in the guinea pig. We have fed filter paper, sawdust and hay respectively, as supplements to an oat diet without averting the appearance of scurvy. Duration of the disease and decline were not appreciably different when these supplements were fed. The addition of 7, 10, or 18 per cent. of paper pulp to a special soy bean diet[1] failed to supply an anti-scorbutic property.

Feeding raw milk in addition to oats induces marked constipation with impaction of feces in the cecum. Animals fed 40 c.c. milk daily showed definite symptoms of scurvy in about a month. As the daily allowance of milk was increased, the symptoms seemed to recede in severity. Yet even when 80 c.c. milk were consumed daily, the animals became very constipated and died; but there were scarcely any signs of scurvy. Autopsy of such a case revealed absence of the typical macroscopic hemorrhages or of fragility of the bones. These observations appear to confirm the findings of Chick, Hume and Skelton,[2] which indicate that a sufficient *amount* of milk fed to guinea pigs will prevent scurvy. Such observations render debatable the hypothesis that

[1] Barnett Cohen, PROC. SOC. EXP. BIOL. AND MED., April 17, 1918, p. 102.

[2] Chick, Hume, and Skelton, *The Lancet*, Jan. 5, 1918.

the experimental scurvy of guinea pigs is attributable to failure of normal intestinal movement.

Preliminary experiments on the nutritive qualities of desiccated vegetables indicate that the *drying* of fresh cabbage does not entirely remove its antiscorbutic property.[1]

181 (1359)

The effect of the respiration of putrid gases upon the growth of guinea pigs.

By C.-E. A. WINSLOW and DAVID GREENBERG.

[*From the Department of Public Health, Yale School of Medicine.*]

Three years ago Mr. G. T. Palmer and the senior author reported here a series of experiments on human subjects conducted by the New York State Commission on Ventilation which indicated that "there are substances present in the air of an unventilated occupied room (even when its temperature and humidity are controlled) which in some way, and without producing conscious discomfort or detectable physiological symptoms, diminish the appetite for food.[2]

A natural assumption would be that odoriferous materials contributed to the stale air by the bodies and clothing of the occupants might produce such an effect upon appetite; and the present study is an attempt to detect a possibly analogous effect of putrid odors of a more intense kind upon the growth of guinea pigs.

A galvanized iron box 4 feet wide, 2 feet deep and 3 feet high was constructed and divided into two equal vertical compartments, each holding two standard animal cages and each provided with a separate glass door. Fresh air to the amount of 1.5 cubic feet per minute for each compartment (amounting to 4 liters per minute per animal) was supplied to the box by a small centrifugal fan, the supply to each section of the box being carefully regulated by dampers. On the course of the branch duct leading from the fan to one section was inserted a chamber in which was placed a pan of fresh moist human or dog feces, so as to produce a strong fecal odor in that section of the box.

[1] See succeeding abstract.

[2] PROC. SOC. EXP. BIOL., Vol. XII, p. 141.

Fifteen different series of tests were conducted with this apparatus during the years 1916, 1917 and 1918, 261 animals in all being used. Young guinea pigs were selected, usually weighing between 150 and 300 grams, and they were kept under the experimental conditions for from 4 to 24 days, being weighed each day. Food supply and other conditions were of course maintained constant in the two compartments.

The results for individual series were somewhat variable, but in general it was evident that the animals exposed to the fecal odor did not grow as fast for the first week as did the controls. Later however they gained more rapidly and by the end of two weeks had generally caught up. This relation appeared in 12 of the 15 series, while in the other 3 no initial check in growth rate was observed.

Calculating the ratio between the weight of the animals exposed to the odor and the weight of the control animals for each day of each series, and then averaging these ratios (which seems a legitimate procedure under the conditions of the experiment) we obtain the general averages tabulated below.

RATIO OF PER CENT. GAIN OF GUINEA PIGS SUBJECTED TO PUTRID ODORS TO CONTROL GUINEA PIGS, BY DAYS.

(Average of 15 Series.)

1	2	3	4	5	6	7	8	9	10	11	12
−10.5	−22.6	−26.8	−27.0	−14.5	−18.7	−20.1	−4.7	−7.6	−8.3	−5.6	−7.4

13	14	15
−2.2	−0.2	0

These results would seem to indicate that the breathing of putrid gases causes a real reduction in the rate of growth of guinea pigs during the first week of exposure; but that this effect is a transitory one, the exposed animals gradually becoming accustomed to the odor and attaining after two weeks a normal weight.

182 (1360)

Studies on the action of certain antiseptics, toxic salts, and alkaloids against the bacteria and protozoa of the intestine of the rabbit

By A. K. BALLS and JOSÉ D. MORAL.

[*From the Department of Bacteriology, Columbia University.*]

The authors believe from theoretical considerations that there is a difference in the susceptibility of bacteria and protozoa to the action of antiseptics. This difference is found to exist between the intestinal bacteria and the intestinal protozoa (*Giardia cuniculi*) of the rabbit, when treated with certain dilutions of chloramine T, brilliant green, "Halazone" and copper sulphate.

In these cases the protozoa are more resistant than the bacteria. Therefore bacterial-free material containing live protozoa is easily obtained.

This behavior does not hold for free living protozoa which are more sensitive to the action of these same antiseptics than the bacteria which accompany them in nature. Free living protozoa are also less resistant when compared with parasitic protozoa.

Increase in temperature greatly increases the effect of the antiseptics, especially against the protozoa.

The authors conclude:

(1) In the intestinal content of the rabbit, it is possible to show that resistance of *Giardia cuniculi* against the action of certain antiseptic substances is markedly greater than the resistance of the intestinal bacteria. It is possible by making use of this difference in resistance to obtain *Giardia* free from all living bacteria, but attempts to subsequently grow them in pure culture have not succeeded. (2) The *Giardia* are more resistant to the action of these toxic substances than are the free living protozoa studied. (3) A technical method has been developed which we believe will be useful in demonstrating the applicability of intestinal antiseptics against both the protozoa and the bacteria of the intestine.

183 (1361)

The influence of cooking and drying cabbage on its antiscorbutic properties for guinea pigs.[1]

By MAURICE H. GIVENS[2] and BARNETT COHEN.

[*From the Sheffield Laboratory of Physiological Chemistry and the School of Medicine, Yale University, New Haven.*]

The experimental scurvy induced in guinea pigs by a special soy bean-milk-yeast-paper pulp-salt diet[3] could be prevented by a daily addition of 10 gm. *raw cabbage* along with the ration. Cabbage *cooked* for thirty minutes at 100° C., subsequently incorporated with the rest of the food, and *dried* at 65–70° C. for two days lost its antiscorbutic power. Cabbage *heated* in an oven for two hours at 75–80° C., then dried at 65–70° C., ground, intimately mixed with the food, and the whole dried further for two days at 65–70° C. exhibited no potency as an antiscorbutic. Cabbage *dried* in a blast of air at *40–52°* C. retained some of its antiscorbutic value in that it delayed markedly the onset of scorbutic symptoms. Furthermore it could be used as a therapeutic agent when the signs of scurvy were recognized early enough.

Diet.	Unmistakble Symptoms Appeared, Days.	Death Ensued, Days.
Soy bean mixture	16–26	23–30.
Soy bean mixture + 10 gm. cabbage	None up to 51 days	
Soy bean mixture + 10 gm. cabbage, "cooked"	14–22	21–34
Soy bean mixture + 1 gm. cabbage, dried 40–52° C.	32–35	39—: two pigs alive at 51 days
Soy bean mixture + 1 gm. cabbage, dried 65–80° C.	14–25	20–35

[1] These experiments were made possible by a grant from the Seessel Research Fund.

[2] Seessel Research Fellow in Physiological Chemistry, Yale University.

[3] B. Cohen, PROC. SOC. EXP. BIOL. AND MED., April 17, 1918, p. 102.

184 (1362)

The prevention of blood clotting by Dakin's sodium hypochlorite solution.

By T. S. GITHENS and S. J. MELTZER.

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

It has been shown by Austin and Taylor that Dakin's solution dissolves necrotic tissue in wounds and washes away blood clots. Sweet has stated that the dressing of wounds with this solution favored hemorrhage from them. We made experiments to determine whether the solution prevented the clotting of blood in vitro and wish to record some of the facts observed.

We found that Dakin's solution of sodium hypochlorite added to blood in vitro was able to prevent clotting. If blood of a dog is drawn directly into Dakin's solution in the proportion of one part of Dakin's to ten of blood, clotting is usually prevented entirely. In a few instances such blood showed the formation of small, soft clots after standing several days. In no case was there any sign of clotting when as much as 1.5 c.c. of Dakin's to 10 c.c. of blood were used, even after standing as much as four weeks. There is a slight difference between the blood of different species; thus cat blood is prevented from clotting by rather smaller amounts than are required for the dog, while rabbit blood requires rather more and guinea-pig blood needs about 4 c.c. for each 10 of blood.

Dakin's solution on standing is said to lose its effectiveness as an antiseptic and its toxicity when injected intraperitoneally. Its power to prevent clotting was found to be reduced to about half in a week and it remained at this point for several weeks. Thus of a certain sample 0.8 c.c. were required to prevent clotting in 10 c.c. of blood when fresh; 1.4 c.c. when 10 days old and 1.5 c.c. when 40 days old.

Blood preserved from clotting by Dakin's solution is not affected by calcium salts but is readily caused to clot by tissue extracts. When the Dakin-blood mixture is fresh the clot formed on addition of tissue extract, forms promptly and becomes firm, but

after the mixture has stood a few days the clot forms more slowly and tends to remain soft. At this time the clot becomes more firm if fresh serum, containing fibrin ferment is added as well as tissue extract. When the mixture has stood for several weeks, clotting can no longer be induced.

185 (1363)

The minimum number of respirations sufficient to maintain life.

By A. L. MEYER and S. J. MELTZER.

[*From the Department of Physiology and Pharmacology, Rockefeller Institute for Medical Research.*]

We have made experiments on the dog to determine how few respirations will maintain life. Both artificial respiration of the ordinary type and the spontaneous respirations of the animal were used for this purpose. We will speak only of the results obtained by the use of artificial respiration.

Ether was used for the exposure of the trachea. A T-shaped glass cannula inserted and firmly tied in the trachea was provided with a respiratory valve, devised by Dr. Meltzer, which in turn was connected with a source of air pressure.

There are two series of experiments. In the first series curare was injected to completely abolish the respiratory movements. We began with two artificial respirations per minute continued for periods varying from 40 to 80 minutes. In all of the 19 experiments the dogs were living at the close of the period. The vagi were intact in 12 experiments; they were cut in 7 experiments. While the volume of air introduced was not measured, it was obviously much below the total lung capacity.

In the second series curare was omitted. A clamp was applied to the trachea below the cannula as a precaution against the entrance of air between the artificial respirations. There were 13 experiments. In 8 the vagi were intact. Of these two survived. Four died in about 25 minutes; two died in about 75 minutes. In 5 experiments the vagi were cut. All died.

186 (1364)

The action of neutral salts on the osmotic pressure and other qualities of gelatin.

By JACQUES LOEB.

[*From The Rocekfeller Institute for Medical Research.*]

Hofmeister and his followers had stated that chiefly the anion of a neutral salt influenced the swelling of gelatin, while the cation had little or no effect. Later on it was stated by colloid chemists that the action of neutral salts on the physical qualities of colloids is the algebraic sum of the opposite action of the two oppositely charged ions of the salt used. Neither of the two statements is correct. The former authors were led to erroneous conclusions by the fact that they always investigated the effect of the salt on the protein in the presence of a high concentration of the salt. Now it happens that the presence of the salt suppresses part of those physical changes in the behavior of the colloids which are caused by the action of the salt upon the protein. The writer avoided this error by using either finely pulverized gelatin or dried pig's bladder, and by washing away the excess of salt after the latter had had a chance to act on the gelatin.

Contrary to the statement current in colloid chemistry, the writer found by this method that (as long as the salts are not used in excessive concentrations) only one of the two ions of the salts acts upon the protein. Common gelatin, as well as gelatin previously treated by a base, is influenced exclusively by the cation of the neutral salt used, while gelatin which had previously been treated with an acid is only influenced by the anion of the salt used. In the former case, treatment of gelatin with salts with monovalent cation (NaCl, Na_2SO_4, etc.) causes an increase in swelling, in viscosity, in the quantity of alcohol required for precipitation, and in osmotic pressure of the gelatin, after the excess of salt is washed away; while when the cation of the salt used is bivalent no such increase is observed. When gelatin, previously treated with an acid, is treated with a neutral salt, only the anion of the latter acts upon the gelatin; the salts with monovalent anion

(NaCl, $CaCl_2$, etc.) causing an increase in the osmotic pressure, viscosity, swelling, etc., while the salts with bivalent anion (sulfates, oxalates, etc.) do not cause such an increase. A very striking proof for the correctness of this statement is found in the fact that the action of salts with monovalent cation and monovalent anion (type NaCl) is equal to that of salts with monovalent cation and bivalent anion (type Na_2SO_4) when the molecular concentration of the former is exactly twice the molecular concentration of the latter; thus showing that only the cation influences the gelatin, while the anion has apparently no effect. For gelatin previously treated with an acid it was found that the salts with monavalent anion and monovalent cation (type NaCl) bring about the same effect as a solution of a salt with bivalent cation and monovalent anion (type $CaCl_2$), when the molecular concentration of the latter salt is exactly one half of that of the former salt, showing that only the anion has any effect in this case.

R. Lillie[1] had stated that the treatment of gelatin with neutral salt causes a lowering of the osmotic pressure. This is not correct and he was misled by the fact that he studied the osmotic pressure of the solution in the presence of the salt, in which case the electrolytic dissociation of the metal gelatinate formed under the influence of the salt is repressed. As soon as the excess of salt is washed away the increase of osmotic pressure of the gelatin solution due to the salt treatment shows itself, provided that the salt used had a monovalent cation. When the cation is bivalent no such increase is found.

The writer is inclined to explain all these phenomena on the assumption that neutral salts act upon proteins, which, like gelatin, are stronger acids than bases, by forming metal proteinates. The anion either does not enter into combination at all with the gelatin or enters into a combination where its influence on the protein is not felt.

When gelatin had been previously treated with an acid, *e. g.*, hydrochloric acid, gelatin chloride or hydrochloride is formed, which dissociates into a positive gelatin ion and a negative chlorion. Neutral salts act upon such gelatin chloride by exchanging their anion with the protein.

[1] Lillie, R. S., *Am. J. Physiol.*, 1907, XX, 127.

This hypothesis explains why the increase in osmotic pressure, viscosity, and the swelling of gelatin caused by the salt does not become noticeable unless the excess of salt is washed away, since the presence of the salt represses the electrolytic dissociation of the gelatin salt formed.

Some of the data on which these conclusions are based have recently been published.[1]

187 (1365)

Studies on salt action. II. The effect of transfer from stronger to weaker salt solutions upon the viability of bacteria in water.

By **C.-E. A. WINSLOW** and **I. S. FALK.**

[*From the Department of Public Health, Yale School of Medicine.*]

In continuation of work previously reported, upon the viability of *B. communis* in salt solutions of various kinds,[2] we have studied the effect of transfer from salt solutions to weaker salt solutions, in order to see if phenomena could be detected analogous to those observed by Loeb[3] and others in the study of the influence of salt solutions upon the swelling of animal membranes and powdered colloids.

B. communis was grown on nutrient agar slants at 37° C. for 16–18 hours. The growth was washed off in pure redistilled water, shaken for five minutes to break up clumps and added in 1 c.c. portions to the electrolyte solutions in which the preliminary treatment was to be accomplished ("primary" solutions), which had been previously warmed to 37° C. The solutions were then shaken for one minute to give a homogeneous suspension, 1 c.c. was withdrawn and agar plates poured. The bottles containing the suspensions were then replaced in the incubator at 37° C. and kept there for 30 minutes (in one case 60 minutes). At the end of this interval plates were again made, 1 c.c. withdrawn and transferred to another bottle containing 99 c.c. of water or salt solution ("secondary" solution). These secondary solution bottles were

[1] Loeb, J., *J. Biol. Chem.*, 1917, XXXI, 343; 1918, XXXIII, 531; XXXIV, 77; 395.

[2] PROC. SOC. EXP. BIOL. AND MED., 1918, Vol. XV, p. 67.

[3] *Jour. Biol. Chem.*, Vol. XXX, p. 343.

then returned to the incubator and kept there throughout the remainder of the experiment. The number of organisms surviving was determined at stated intervals. The counts were made on standard agar after 24 hours incubation at 37° C.

All the reagents used in these experiments were of the same high purity as those which were employed in the experiments reported in our earlier communication. All the glassware had been very carefully cleaned with chromic acid oxidizing mixture, rinsed in tap water and in distilled water.

Table I shows the result of transfer from 0.1 isotonic NaCl solution to 0.001 isotonic NaCl (1 c.c. of the first solution containing the bacteria to 99 c.c. of sterile distilled water), from 0.1 isotonic NaCl to another bottle of the same solution, and from 0.1 isotonic $CaCl_2$ to another bottle of the same solution. Three independent experiments are cited in each group. In each of these three series of experiments the reduction in bacterial numbers was relatively small in 9 hours although in the case of the $CaCl_2$ the normal toxicity of this salt manifested itself to a slight degree. In each case, of course, there is a decrease in actual count in the transfer to about 1 per cent. of the original number present, but this is the result of the dilution and does not imply the death of the bacteria.

The transfer from 0.1 isotonic $CaCl_2$ to 0.001 isotonic $CaCl_2$ as shown in Table II is accompanied on the other hand by a rapid destruction of bacteria so that after 8–9 hours only a few hundred bacteria are left out of hundreds of thousands.

TABLE I.

VIABILITY OF *B. communis* TRANSFERRED FROM A STRONG TO A WEAK SODIUM SOLUTION AND FROM SODIUM AND CALCIUM SOLUTIONS TO SOLUTIONS OF THE SAME STRENGTH.

		Time after Seeding.		Time after Transfer.			
		1 Min.	30 Min.	120 Min.	240 Min.	360 Min.	540 Min.
0.1 isotonic	Bacteria	29,600,000	29,700,000	280,000	270,000	260,000	270,000
NaCl to 0.001	per	35,500,000	32,500,000	285,000	240,000	235,000	210,000
isotonic NaCl	c.c.	33,500,000	30,000,000	215,000	195,000	195,000	180,000
0.1 isotonic	Bacteria	35,500,000	32,500,000	370,000	312,000	310,000	240,000
NaCl to 0.1	per	33,500,000	30,000,000	260,000	245,000	255,000	210,000
isotonic NaCl	c.c.	33,500,000	30,000,000	255,000	230,000	260,000	230,000
0.1 isotonic	Bacteria	26,500,000	26,500,000	300,000	300,000	240,000	170,000
$CaCl_2$ to 0.1	per	29,200,000	28,300,000	200,000	170,000	70,000	40,000
isotonic $CaCl_2$	c.c.	33,500,000	26,500,000	295,000	255,000	210,000	170,000

TABLE II.

VIABILITY OF *B. communis* TRANSFERRED FROM A STRONG TO A WEAK CALCIUM SOLUTION.

		Time after Seeding.			Time after Transfer.											
		1 Min.	30 Min.	60 Min.	15 Min.	30 Min.	60 Min.	105 Min.	120 Min.	240 Min.	280 Min.	310 Min.	360 Min.	460 Min.	490 Min.	540 Min.
0.1 isotonic $CaCl_2$ to .001 isotonic $CaCl_2$	Bacteria per c.c.	86,000,000	81,000,000		500,000	480,000	65,000		30,000			650			110	
		26,500,000	26,500,000						260,000	80,000			32,500			<1,000
		63,000,000		68,000,000	540,000	520,000	360,000	150,000			1,900			150		

In Loeb's colloid studies it was a transfer from NaCl to more dilute NaCl or water which produced a marked increase in swelling. In the case of the viability of the bacteria it is a transfer from $CaCl_2$ to a very dilute solution which causes a profound effect. Whether the death of the bacteria in the latter case is due to the fact that compounds are formed in the cell wall or protoplasm which hinder the elimination of toxic waste products of cell metabolism or whether such compounds favor the ingress of water which causes deadly hydrolyses (which Phelps has suggested as the lethal factor in similar cases), or whether such compounds favor the loss of necessary constituents of the cell to the water outside, we are not prepared to say. Experiments are now being conducted to throw light upon this point. It seems clear in any case from the conditions of the experiment that the effect is not due to any direct toxicity of the salt but to some change which it produces in the rate of exchange between the inside of the cell and its environment.

188 (1366)

Experimental tri-nitro-toluene poisoning.

By **RUDOLPH KRAMER** and **HAROLD MEIERHOF** (by invitation).

[*From the Pathological Department, College of Physicians and Surgeons.*]

In an attempt to produce experimentally in dogs a state of poisoning by tri-nitro-toluene analogous to the condition recently observed among munition workers in England and America, the following methods of administering the poison have been employed: (1) Feeding by mouth (TNT in butter); (2) skin inunction (TNT in lard); (3) subcutaneous injections (TNT in olive oil); (4) intravenous injections (TNT in acetone); (5) intraperitoneal injections (TNT in albolene). Only the first three methods have proven satisfactory. Intravenous injection of any considerable quantity of an acetone solution causes immediate death, probably from a precipitation of the TNT in the blood stream, and consequent pulmonary embolism. The toxic action of the acetone, too, may be a factor. Negative results with intraperitoneal injections of an albolene solution were probably due to faulty absorption.

Feeding, inunction, and subcutaneous injections have resulted regularly in a more or less chronic state of poisoning ending in death. The rapidity with which toxic symptoms appear and the duration of life depend apparently on both the quantity of poison given and the frequency and method of administration. Skin inunction has so far given most constant results.

Symptoms observed: (1) Vomiting; seen only in feeding cases; apparently due to direct irritation of the stomach by the poison; (2) diarrhea; frequently present, its occurrence is not related to any particular method of administration; (3) depression, surliness, weakness, and emaciation, very marked in later stages; (4) leucocytosis. In one case a slight relative increase in lymphocytes, polychromasia, and nucleated R. B. C. (megaloblasts) was noted.

Autopsy findings: Heart, lungs, and gastro-intestinal tract negative. Liver showed grossly either no change or the picture of a moderate chronic passive congestion with lobules dark red in center and pale in periphery. Isolated yellowish, opaque nodules were sometimes seen scattered throughout the organ. Microscopically, a moderate central degeneration of the liver cells with congestion of the capillaries about the efferent veins was found. Fatty changes were demonstrable here and there, but the lesion was not uniformly distributed. In one case there were nodules of liver cells comprising several lobules, showing extreme fat accumulation. In the bone marrow, lymph nodes, and spleen there was an increased amount of blood pigment lying free in the tissues and in large phagocytic cells.

The outstanding feature of the autopsy findings is the absence of lesions which would explain the death of the animal. The changes in the liver, while definite and perhaps significant, are not comparable to those found in cases of toxic jaundice in human beings, where the destruction of liver tissue is as extreme as in acute yellow atrophy.

189 (1367)

The toxic action of nitrobenzene, with special reference to the cerebellum.

By **M. DRESBACH** and **W. L. CHANDLER.**

[*From the Department of Physiology and Biochemistry, Cornell Medical College, Ithaca, N. Y.*]

The toxic action of nitrobenzene has been comparatively little studied in an experimental way. The investigations here reported have been in progress about two years. Dogs, cats, rabbits, guinea pigs, white rats and mice, hens, pigeons, frogs, and certain insects and blood parasites have been used. The animals have in all cases been exposed to air saturated with the vapor of the nitrobenzene at various temperatures in a special metal box of forty cubic feet capacity, and so arranged that good ventilation was insured. The periods of exposure varied from one to twenty-four hours, or even longer, as in case of rabbits and guinea pigs; with other animals the exposure was usually much shorter. The effects obtained vary with the type of animal somewhat. In dogs and birds the nervous system disturbances predominate, with lighter dosage, while in the other mammals used blood changes are more prominent, especially in severe poisoning. The details of these variations will be published later. At this time we give only the central nervous system reactions. These are of a type that are associated with cerebellar disturbance especially. Thus, in dogs an early asthenic condition in the limb and neck muscles, staggering gait, typical cerebellar nystagmus, unequal pupils, "circus" movements may be seen. In birds a body attitude and rotating motion of the head are strikingly similar to those in birds with cerebellar lesions. The animals may recover, sustain permanent disturbance of muscular coördination, or die. Symptoms develop at any time up to three or four days after exposure to the vapor.

Histological study of various parts of the brain and cord revealed remarkable chromatolytic changes, apparently confined to the Purkinje cells of the cerebellum, in all animals showing

disturbed coördination. The chromatolysis in these cells seems to be roughly proportional to the intensity of symptoms. In general, the cytoplasm and nuclei swell, often to several times the normal size, the tigroid substance becomes faint and collected around the nucleus, or disappears altogether. Often the entire cell shrinks and disappears completely. In one dog with disturbed motor function resulting from exposure to the vapor several months before the sectioning of the brain no Purkinje cells at all could be found in some parts of the cerebellum. All signs pointed to a permanent lesion. A hen showed a similar permanent effect. In these two cases the fatigued or weakened muscle activity in the legs resembled closely the type said to be associated with lesions of certain cerebellar tracts.

Since the Purkinje cell axones are the only efferent paths from the cerebellar cortex and since the nitrobenzene attacks these cells especially, if not selectively, the results are suggestive. While we cannot claim a specific and direct effect of nitrobenzene on these cells, as far as chromatolysis may indicate it the action is not much in evidence, if at all, in other parts of the nervous system.

Animals poisoned in this way illustrate nicely cerebellar disorders for teaching purposes. They also show in a striking way an instance of delayed development of toxic action of a substance stored in the body. This latent period in nitrobenzene poisoning forms one of its most interesting features and is being studied by the writers. Other interesting problems have been opened up by the work.

190 (1368)

A method for the simultaneous fractional analyses of gastric and duodenal contents.

By MAX KAHN.

[*From the Department of Laboratories, Beth Israel Hospital, New York.*]

It is possible to study simultaneously the duodenal and gastric secretions by the following method: An Einhorn tube is passed into the duodenum of the patient, using the technic of Einhorn. Next

morning a Rehfuss tube is inserted into the stomach of the patient. The patient is then given an Ewald test meal, and the gastric and duodenal contents removed simultaneously at varying intervals of time. The extractions are usually made every fifteen minutes for a period of two and a half or three hours. The gastric contents are analyzed for the acid secretions and the enzymes. The duodenal contents are analyzed for the various enzymes. The results are charted in the form of a curve.

A gastro-duodenal tube has been devised which obviates the necessity of passing two tubes. This tube is composed of two compartments—one ending ten inches above the duodenal opening. The tube bifurcates at its free end, and the openings are distinctly labelled G and D to indicate the opening leading to the stomach and to the duodenum.

The clinical and physiological results of the study of the gastric and duodenal secretions by this method will soon be reported.

191 (1369)

Studies on the metabolism of cells in vitro. The toxicity of dipeptids for embryonic chicken cells.

By MONTROSE T. BURROWS and CLARENCE A. NEYMANN.

[*From the Pathological Laboratory, Washington University Medical School, St. Louis, Mo., and the Laboratory of Internal Medicine, Henry Phipps Psychiatric-Clinic, The Johns Hopkins University, Baltimore.*]

In a previous paper[1] we have shown that peptone prepared from the yolk of egg is non-toxic for growing chicken cells even when added in considerable concentration to the medium. Egg yolk digested to the point of crystallizing out an *a*-amino acid is toxic. We tested a large number of *a*-amino acids and have found that all are toxic for the cells. In low dilution they stimulate the contraction of heart muscle fragments but did not affect the growth. In higher dilution they inhibit the growth of cells completely and killed.

Having established this fact it became of interest to study the

[1] *Jour. Exp. Med.*, 1917, Vol. XXV., pp. 93–108.

effect of dipeptids in a similar manner. Leucyl-glycin and carnosin have been tested. The leucyl-glycin was a synthetic product. The carnosin was obtained from muscle. It was prepared by Dr. D. W. Wilson.

Both of these substances are toxic in large doses. Their toxicity is not as marked, however, as is that of the *a*-amino acids. The leucylglycin is more toxic than the carnosin.

During the course of these experiments we prepared and tested peptones prepared from beef. These were found not to affect in any way the growth of the cells.

192 (1370)

A note on the healing in and migration of foreign bodies in the animal body.

By **L. S. N. WALSH** and **LEO LOEB.**

[*From the Department of Comparative Pathology, Washington University, St. Louis.*]

We have made some observations on the difference in the behavior of different species of animals towards foreign bodies on which we wish to report very briefly.

If we apply very thin wires of copper around the neck of white rats in such a way that apparently they do not exert any marked pressure on the skin, these wires heal in at the ventral aspect of the neck, but not at the dorsal and lateral aspects, in a considerable number of cases, after they have at first produced some ulceration. About four weeks after the onset of the ulceration the skin begins to heal over the wire and soon the wire is buried deep in apparently normal tissue. Gradually the wire may migrate deeper into the tissues of the ventral parts of the neck and in one case it was found resting on the trachea, the tissues covering it being apparently quite normal. Thus the migration of foreign bodies can be imitated experimentally in certain cases.

In a large number of white mice and in three guinea pigs wires which had been applied in a similar manner, or even somewhat more tightly, did not heal in, but led to a transitory, more or less, superficial ulceration and scab formation. After some time such ulcers

may heal, only to form again, in case the wire should continue to irritate the skin.

Microscopically we find in the rat in the first stage, that of ulceration, the base of the ulcer formed by fibrous or hyaline tissue which is infiltrated by fibroblasts. The wire is seen lying at the base of the ulcer surrounded by necrotic tissue and polynuclear leucocytes. On the whole, the capillaries run radially towards the surface of the ulcer, while the fibrous tissue and the fibroblasts are arranged in a direction more or less parallel to the surface of the ulcer. At the side of the ulcer near the tip of the regenerating epithelium, the growing connective tissue is more cellular and less fibrous and the capillaries are more dilated and a greater influx of polynuclear leucocytes is observed than at the base of the ulcer; at the latter place the pressure is greater and this may be responsible for the greater production of dense fibrous tissue and the poorer development of capillaries.

In cases in which the wire healed in we find it in later periods embedded in a canal which is surrounded by a small zone of necrotic tissue and polynuclear leucocytes. This zone is followed by an area of dense fibrous tissue, surrounding the wire on the whole more or less concentrically (although some deviations may occur), while the capillaries run more or less in a radial direction towards the wire; however, at some places they may adapt themselves to the concentric course of the fibrous tissue. Certain deviations from this arrangement seem to occur. Mechanical factors, pressure and pull, and a stereotropic reaction, determine probably essentially the direction in which the fibroblasts are arranged, and pressure as well as certain chemical factors cause probably the transformation of the fibroblastic into fibrous tissue.

The blood capillaries evidently do not respond to the same degree to the mechanical factors which determine the movements of the fibroblasts; they often run in a direction approximately radially to the lines of pressure and often continue to follow this direction at a time when the connective tissue has assumed an arrangement parallel to the lines of pressure and at right angles to the blood vessels. We may point out that from a teleological point of view, the difference in the reaction of connective tissue and capillaries appears as a useful adaptation to the requirements

of tissue growth. The radial growth of the capillaries, in contradistinction to the concentric growth of the connective tissue makes possible the nourishment and persistence of the newly formed tissue, and thus the definite organization of dead material.

193 (1371)

Experiments on antiscorbutics. Report of an antiscorbutic for intravenous use.

By ALFRED F. HESS and LESTER J. UNGER.

[*From the Bureau of Laboratories, Department of Health, N. Y. City.*]

At the last meeting of this Society we presented a paper on guinea-pig scurvy, which had been induced by a diet of oats, hay, and water. In the present paper we wish to report some protective and curative experiments on pigs which were fed on this diet, as well as on the use of various antiscorbutics in the scurvy of infants.

Guinea pigs developed scurvy on the above diet in spite of a daily per capita allowance of five grams of dried vegetables. Three lots of vegetables were use on three groups of pigs: (1) a commercial preparation of mixed vegetables; (2) carrots dried at room temperature last summer; (3) carrots rapidly dried a few weeks previously at a temperature of about 130° F. All the pigs in each group developed scurvy, those of group 3 developing the disease somewhat later than the others. A watery decoction of orange peel was able to protect against scurvy. A similar preparation, made from orange peel that had been dried at room temperature a few months, retained only mild antiscorbutic properties.

Orange juice proved to be a most effective antiscorbutic in very small amounts. If, however, it was kept in the refrigerator for about three months it lost considerable of its potency, the pigs to which it was fed failing to gain normally. Orange juice which had been subjected in an autoclave to 110° C., at ten to fifteen pounds pressure, although antiscorbutic, did not enable pigs to gain in weight as did the unheated juice. Orange juice that was

extracted with absolute or 95 per cent. alcohol, possessed anti-scorbutic qualities, whereas the residue was of no value in this respect. Neutralized orange juice, given subcutaneously to a group of guinea pigs, also failed in this respect. "Artificial orange juice," made up according to McCollum's formula, composed of the various salts, citric acid, and sucrose in the proportions which they are found in the natural juice, was given to numerous pigs, but was ineffective in protecting them against scurvy.

"Artificial orange juice" was likewise tried in the treatment of two infants suffering from scurvy. In both it failed absolutely to ameliorate the symptoms, which yielded rapidly to natural orange juice.

We wish to report especially that *orange juice may be given intravenously*, after it has been boiled, and made slightly alkaline to litmus, by the addition of normal sodium hydrate just before using, and that such injections produce no untoward reaction, and have a marked curative effect. This intravenous therapy is of interest from a practical standpoint, as its action has been found to be most rapid, and its dosage small; it is therefore especially indicated where the symptoms are urgent. It is also of theoretical interest in a consideration of the pathogenesis of scurvy, as it is the first time that this disorder has been cured by a therapy which was not introduced by way of the alimentary tract.

194 (1372)

A delicate method of determining invert activity.

By **C. K. WATANABE** and **V. C. MYERS.**

[*From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.*]

A delicate method of estimating invert activity (sucrase) would be of advantage in connection with a number of physiological problems. A substitution of one per cent. cane sugar for the one per cent. soluble starch or glycogen, employed by Myers and Killian,[1] in the estimation of the diastatic activity of the blood, furnishes a very delicate method for sucrase.

[1] Myers and Killian, *Jour. Biol. Chem.*, 1917, XXIX, 179.

The technic of the method is as follows: To 8 c.c. of water in a 50 c.c. centrifuge tube is added 1 c.c. of the extract to be examined, and the solution warmed to just 40° C. in a water bath with thermostat attachment. One c.c. of one per cent. cane sugar is now added and incubation carried out for 30 minutes. The solution is rapidly cooled in cold water and 0.5 to 1.0 gram of dry picric acid added, thoroughly mixed, centrifuged and filtered. The inverted sugar is now estimated colorimetrically in 3 c.c. portions as already described.[1]

One must not let the sucrose stand for a long time in contact with the picric acid, owing to the hydrolytic action of the picric acid.[2] For this reason it might be of advantage to employ sodium picrate, as recently recommended by Benedict.[3]

Utilizing the above method we have been unable to satisfactorily demonstrate sucrase in human blood or that of rabbits. With one per cent. yeast extract there was 60 per cent. inversion in one-half hour, *i. e.*, 6 of the 10 mg. were inverted, while a one per cent. yeast extract in human blood gave a 54 per cent. inversion. With a 10 per cent. extract of the mucous membrane of rabbit's intestine figures of 13.2 and 12.8 per cent. were obtained. Controls were all negative.

195 (1373)

The phosphate and calcium content of serum in the condition of guanidine tetany.

By C. K. WATANABE.

[*From the Laboratory of Pathological Chemistry, School of Medicine, and The Sheffield Laboratory of Physiological Chemistry, Yale University, New Haven.*]

In the altered metabolism in parathyroidectomized dogs, Greenwald[4] found a marked decrease in the elimination of phosphate in the urine together with a distinct retention in the blood.

[1] Myers and Bailey, *Jour. Biol. Chem.*, 1916, XXIV, 147.

[2] Rose, these PROCEEDINGS, 1917, XX, 16.

[3] Benedict, *Jour. Biol. Chem.*, 1918, XXXIV, 203.

[4] Greenwald, I., *Am. Jour. Physiol.*, 1911, XXVIII, 103.
Greenwald, I., *J. Biol. Chem.*, 1913, XIV, 363; Ibid., 1913, XIV, 369.

It has been established that the lack of parathyroid is accompanied by increased elimination of ammonia, decreased acid excretion and the lowering of the hydrogen ion concentration. The same conditions are manifested after the administration of guanidine hydrochloride.[1] The increase in guanidine nitrogen was found by Koch[2] and Burns and Sharpe[3] in experimental parathyroid tetany and also in idiopathic tetany. In a recent communication[4] we reported that the above-mentioned phenomena were an accompaniment of the acidosis produced by guanidine administration and also suggested that phosphates might be retained in the body to neutralize the acids formed by the muscular activity.

Another fact that in parathyroid tetany, the calcium content of the tissues was decreased was reported by Sabbatani[5] and Quest[6] and by many other investigators. In this country MacCallum and Voegtlin[7] confirmed the above statements and also reported a marked reduction in the blood calcium caused by the increased output in the urine and feces. It, therefore, seemed advisable to inquire into the phosphate and calcium changes in the blood in guanidine tetany and to compare these changes with those occurring in parathyroid tetany in order to decide whether the increase in guanidine nitrogen is the only cause of this phenomenon or whether other factors are involved in the production of parathyroid tetany.

Marriott and Howland's methods were employed in this investigation. Rabbits were used and two samples of blood were drawn from the jugular vein; one before the subcutaneous administration of a sublethal dose of guanidine hydrochloride and the other after—about 10 to 48 hours after the injection of the drug. It is necessary to wait for the second sample of blood until the acidosis is well developed, though most animals do not live long after this condition becomes severe. Since animals usually died at midnight or early in the morning, the second blood sample

[1] Watanabe, C. K., *J. Biol. Chem.*, 1918, XXXIV, 51.
[2] Koch, W. F., *J. Biol. Chem.*, 1912, XII, 313; Ibid., 1913, XV, 43.
[3] Burns, D. and Sharpe, J. S., *Quart. J. Exp. Physiol.*, 1916, X, 345.
[4] Watanabe, C. K., *J. Biol. Chem.*, 1918, XXIV, 65.
[5] Sabbatani, "Rivist. sperim. di frematria," 1901 (quoted by Quest).
[6] Quest, R., *Jahrb. f. Kindelheinlkunde*, 1905, LXI, 114.
[7] MacCallum, W. G. and Voegtlin, C. J., *Exp. Med.*, 1909, XI, 118.

could not be taken and thus comparisons of the normal with the tetanic samples were impossible.

From the comparison in the result it is clear that after guanidine administration the phosphorus is markedly increased, in some cases five times above the normal. The calcium shows no marked change, though there is a tendency for it to decrease as the phosphate increases. In normal conditions, rabbits with albumin in the urine show a rather higher content of phosphate than those which have no albumin.

In consideration of the above experiments we may say that the phosphate content of the serum in guanidine tetany is markedly increased, but that the reduction of calcium is rather doubtful. The small number of experiments does not warrant a decision on this important phenomenon. The experiments are being continued and further results will be reported later.

196 (1374)

An oxidation product of creatine.

By L. BAUMANN and THORSTEN INGVALDSEN.

From the Chemical Research Laboratory, Department of Internal Medicine, State University of Iowa, Iowa City.

Mercuric acetate in watery solution oxidizes creatine to α-methylguanidoglyoxylic acid ($NH_2.C(:NH).N(CH_3)CO.COOH$). This compound, which was isolated in pure form, evidently precedes methylguanidine oxalate which was obtained by Dessaignes[1] many years ago by heating creatine with mercuric oxide. Dakin[2] obtained glyoxylic acid upon oxidizing creatine with hydrogen peroxide. The stages of oxidation of creatine may, therefore, be expressed as follows:

$$NH_2.C(:NH).N(CH_3).CH_2COOH \xrightarrow{O} NH_2.C(:NH).N(CH_3).CHOH.COOH \xrightarrow{O}$$

$$NH_2.C(:NH).N(CH_3).CO.COOH \xrightarrow{H_2O} NH_2C(:NH).NH(CH_3).COOH.COOH.$$

[1] Dessaignes, M., *Compt. rend. Acad.*, 1854, XXXVIII, 839.

[2] Dakin, H. D., *J. Biol. Chem.*, 1905–06, I, 271.

In the well-known Engeland[1] process for the separation of the bases of muscle extract, precipitation with mercuric chloride and sodium acetate is used. After several days the precipitate is collected and heated with dilute hydrochloric acid, then decomposed with hydrogen sulphide. Under these conditions the possibility of creatine oxidation, hydrolysis of the resulting compound and liberation of methylguanidine, must be considered.

197 (1375)

Concerning carnosine and its synthesis.

By L. BAUMANN and THORSTEN INGVALDSEN.

[*From the Chemical Research Laboratory, Department of Internal Medicine, State University of Iowa, Iowa City.*]

The chemical configuration of the muscle extractive, carnosine, has been determined in two ways: (1) By deamination with nitrous acid, hydrolysis of the resulting deaminocarnosine and isolation of one of the cleavage products. (2) By its synthesis.

The hydrolysis of deaminocarnosine gave a 70 per cent. yield of histidine.

The synthesis was effected by the interaction of beta iodopropionyl chloride and histidine, followed by amination of the resulting product. The analyses, optical rotation, melting point and crystal form of the synthetic and natural products were identical. A mixture of both substances gave the same melting point as either component.

It is evident that beta alanyl-histidine correctly expresses the constitution of carnosine.

Carnosine is not hydrolyzed by muscle or liver extract.

[1] Engeland, R., *Z. Unters. Nahr. Genussmittel*, 1908, XVI, 658.

RECAPITULATION OF THE NAMES OF THE AUTHORS AND OF THE TITLES OF THE COMMUNICATIONS. VOLUME XV.

Abramson, H. L.

1345. Immunization of monkeys against poliomyelitis.

Addis, T.

1333. [with **A. E. Shevky.**] Sources of error in the estimation of dextrose by the calorimetric picrate method.

Amoss, Harold L.

1311. [with **Frederick Eberson.**] Therapeutic experiments with Rosenow's antipoliomyelitic serum.

Atchley, Dana W.

1338. Renal action in acute nephritis.

Auer, John.

1284. [with **Israel S. Kleiner.**] Morphin hyperglycaemia as a test for pancreatic deficiency.

1307. [with **S. J. Meltzer.**] Lantern slide demonstration of the effect of magnesium sulphate upon tetanus.

1350. Generalized analgesia in cats after exposure to a war-gas, $(CH_3)_2SO_4$.

1351. Localized pulmonary edema in cats after inhalation of a war-gas, $(CH_3)_2SO_4$.

Austin, James H.

1303. See **Taylor, Herbert D.**

1310. See **Cullen, Glenn E.**

Balls, A. K.

1360. [with **José D. Moral.**] Studies on the action of certain antiseptics, toxic salts and alkaloids against the bacteria and protozoa of the intestine of the rabbit.

Baumann, L.

1374. [with **Thorsten Ingvaldsen.**] An oxidation product of creatine.

1375. [with **Thorsten Ingvaldsen.**] Concerning carnosine and its synthesis.

Bergeim, Olaf.

1290. The determination and significance of intragastric conductance.

1291. See **Halverson, John O.**

Burnett, Theodore C.

1334. Does the liver secrete a catalase accelerator?

Burrows, Montrose T.

1369. [with **Clarence A. Neymann.**] Studies on the metabolism of cells in vitro. The toxicity of dipeptids for embryonic chicken cells.

Chandler, W. L.

1367. See **Dresbach, M.**

Clowes, G. H. A.

1352. On the electrical resistance and permeability of tumor tissues.

1353. On the action exerted by antagonistic electrolytes on the electrical resistance and permeability of emulsion membranes.

Cohen, Barnett.

1348. Observations on the production of experimental scurvy in guinea pig.

1358. [with **Lafayette B. Mendel.**] Diet and roughage in relation to the experimental scurvy of guinea pigs.

1361. See **Givens, Maurice H.**

Coleman, Warren.

1341. Asynchronism of the respiratory movements in lobar pneumonia.

Connet, Helene.

1289. See **Salant, William.**

Coombs, Helen C.

1319. [with **F. H. Pike.**] The rôle of afferent impulses in the control of respiratory movements.

Cullen, Glenn E.

1310. [with **James H. Austin.**] A note on the preparation of Dakin's hypochlorite solution.

Dresbach, M.

1367. [with **W. L. Chandler.**] The toxic action of nitrobenzene with special reference to the cerebellum.

Eberson, Frederick.

1311. See **Amoss, Harold L.**

Ellefson, Lillian J.

1356. [with **Carl L. A. Schmidt.**] On serum proteins.

Epstein, Albert A.

1339. The application of the Auer-Kleiner morphine test in human diabetes.

Erdmann, Rhoda.

1344. Production of transplantable growth.

Falk, I. S.

1326. See **Winslow, C.-E. A.**

1365. See **Winslow, C.-E. A.**

Falk, K. George.

1301. [with **Kanematsu Sugiura.**] The two forms of glycine.

Friesner, I.

1317. See **Strauss, I.**

Gies, William J.

1287. [with collaborators.] Studies of dental caries, with special reference to internal secretions in their relation to the development and condition of dental enamel.

Githens, T. S.

1328. [with **S. J. Meltzer.**] Studies in experimental shock.

1362. [with **S. J. Meltzer.**] The prevention of blood clotting by Dakin's sodium hypochlorite solution.

Givens, Maurice H.

1325. Studies in calcium and magnesium metabolism. Further observations on the effect of acid and dietary factors.

1347. The composition of dried vegetables with special reference to their nitrogen and calcium content.

1361. [with **Barnett Cohen.**] The influence of cooking and drying cabbage on its antiscorbutic properties for guinea pigs.

Greenberg, David.

1359. See **Winslow, C.-E. A.**

Greenberg, J. P.

1313. See **Macht, David I.**

1323. See **Macht, David I.**

Halverson, John O.

1291. [with **Henry K. Mohler** and **Olaf Bergeim.**] The calcium content of the blood serum in certain pathological conditions.

Hess, Alfred F.

1332. See **Torrey, J. C.**

1336. [with **Lester J. Unger.**] Experiments on the scurvy of guinea pigs.

1371. [with **Lester, J. Unger.**] Experiments on antiscorbutics. Report of an antiscorbutic for intravenous use.

Hewlett, A. W.

1292. Circulatory effects of tyramin.

Hoskins, E. R.

1349. [with **M. M. Hoskins.**] Further experiments with thyroidectomy in Amphibia.

Hoskins, M. M.

1349. See **Hoskins, E. R.**

Hulton-Frankel, Florence.

1304. [with **Katherine MacDonald.**] Differentiation of typhoid, paratyphoid *A* and *B* by means of a dextrin-inosite medium.

Ingvaldsen, Thorsten.

1374. See **Baumann, L.**

1375. See **Baumann, L.**

Isaacson, V. I.

1294. See **Janney, N. W.**

1327. See **Janney, N. W.**

Isaacs, S.

1313. See **Macht, David I.**

1323. See **Macht, David I.**

Janney, N. W.

1294. [with **V. I. Isaacson.**] A blood sugar tolerance test.

1327. [with **V. I. Isaacson.**] Protein sparing by glucose in experimental diabetes.

Kahn, Max.

1331. Sulpho-conjugation as a test of hepatic function.

1368. A method for the simultaneous fractional analyses of gastric and duodenal contents.

Kast, Ludwig.

1283. [with **V. C. Myers** and **Emma L. Wardell.**] The estimation of cholesterol in blood.

1343. [with **Emma M. Wardell.**] The urea content of the blood.

Killian, John A.

1296. The influence of operative anesthesia and alkali therapy on the diastatic activity and sugar of human blood.

Kleiner, Israel S.

1284. See **Auer, John.**

1286. [with **S. J. Meltzer.**] The effect of painting the pancreas with adrenalin on glycaemia and glycosuria.

1335. The rate of dialysis of diabetic blood-sugar.

Kolmer, John A.

1342. See **Weiss, Charles.**

Kramer, Rudolph.

1366. [with **Harold Meierhof.**] Experimental trinitrotoluene poisoning.

Krigel, H.

1298. A new method of tissue culture for accurate and rapid measurements of the growth.

Kuttner, Theodore.

1340. A method for determining minute quantities of nitrogen in nitrogenous substances.

Lambert, Robert A.

1354. [with **S. S. Samuels.**] The relationship of the leucocyte count and bone-marrow changes in acute lobar pneumonia.

Lathrop, A. E. C.

1330. See **Loeb, Leo.**

Laughlin, H. H.

1357. The dynamics of cell-division.

Levene, P. A.

1299. Uridin and cytidin-phosphoric acid.

1305. [with **C. J. West.**] Hydrolecithin and its bearing on the constitution of cephalin.

Levin, Isaac.

1300. [with **M. Levine.**] The influence of X-Rays on the development of the crown gall.

Levine, M.

1300. See **Levin, Isaac.**

Loeb, Jacques.

1315. The chemical basis of morphological polarity in regeneration.

1364. The action of neutral salts on the osmotic pressure and other qualities of gelatin.

Loeb, Leo.

1330. [with **A. E. C. Lathrop.**] The effect of continued inbreeding on the tumor rate in mice.

1370. See **Walsh, L. S. N.**

MacDonald, Katherine.

1304. See **Hulton-Frankel, Florence.**

MacDougal, D. T.

1314. The relation of growth and swelling of plants and biocolloids to temperature.

Macht, David I.

1302. On the absorption of apomorphin and morphin through unusual channels.

1312. On the comparative absorptive power for drugs of the bladder and urethra (male).

1313. [with **S. Isaacs** and **J. P. Greenberg.**] On the influence of some opiates and antipyretics on the field of vision.

1323. [with **S. Isaacs** and **J. P. Greenberg.**] On the influence of some antipyretics on the neuro-muscular coördination test of "tapping."

1324. On the relation of the chemical structure of the opium alkaloids to their effect on smooth muscle, and the discovery of a new therapeutic agent as a consequence thereof.

Meierhof, Harold.

1366. See **Kramer, Rudolph.**

Meltzer, S. J.

1286. See **Kleiner, I. S.**

1307. See **Auer, J.**

1321. See **Wollstein, Martha.**

1328. See **Githens, T. S.**

1362. See **Githens, T. S.**

1363. See **Meyer, A. L.**

Mendel, Lafayette B.

1329. [with **Thomas B. Osborne.**] Further observations on the nutritive factors in animal tissues.

1358. See **Cohen, Barnett.**

Meyer, A. L.

1363. [with **S. J. Meltzer.**] The minimum number of respirations sufficient to maintain life.

Mohler, Henry K.

1291. See **Halverson, John O.**

Moral, Jose D.

1360. See **Balls, A. K.**

Moore, A. R.

1297. Acid development as the result of injury in nervous tissue.

Morgan, T. H.

1285. Demonstration of the effects of castration on Seabright cockerels.

Myers, V. C.

1283. See **Kast, Ludwig.**

1372. See **Watanabe, C. K.**

Neun, D. E.

1318. See **Sherman, H. C.**

Neymann, Clarence A.

1369. See **Burrows, Montrose T.**

Olmstead, Miriam

1337. Types of pneumococci found in the mouths of surgical cases before operation.

Ophüls, W.

1355. [with **Elmer W. Smith.**] Anatomical changes produced by repeated intravenous injections of streptococci in rabbits.

Osborne, Thomas B.

1329. See **Mendel, Lafayette B.**

Park, William H.

1346. Persistence of immunity following toxin-antitoxin injections.

Pike, F. H.

1319. See **Coombs, Helen C.**

Porter, Eugene L.

1322. Changes in reflex thresholds following experimental shock from intestinal manipulation.

Riddle, Oscar.

1320. A case of hereditary ataxia (?) in pigeons.

Ringer, A. I.

1309. Demonstration of blood from an extreme case of lipemia in diabetes mellitus.

Rose, Anton R.

1295. The inversion and determination of cane sugar.

Salant, William.

1288. [with **A. M. Swanson.**] Diet and renal activity in tartrate nephritis.

1289. [with **Helene Connet.**] Experiments with an isomer of caffein.

Samuels, S. S.

1354. See **Lambert, Robert A.**

Schmidt, Carl L. A.

1356. See **Ellefson, Lillian.**

Sherman, H. C.

1318. [with **D. E. Neun.**] Amylase and protease action of some pancreas preparations.

Shevky, Ahmed E.

1293. Absence of urea-splitting ferments in the animal tissues.

1333. See **Addis, T.**

Smith, Elmer W.

1355. See **Ophüls, W.**

Stout, A. B.

1316. Experimental studies of self-incompatibilities in fertilization.

Strauss, I.

1317. [with **I. Friesner.**] A demonstration of cerebellar and cerebral lesions in dogs.

Sugiura, Kanematsu.

1301. See **Falk, K. George.**

Swanson, A. M.

1288. See **Salant, William.**

Taylor, Herbert D.

1303. [with **J. Harold Austin.**] The solvent action of antiseptics on necrotic tissue.

Torrey, J. C.

1332. [with **Alfred F. Hess.**] The relation of the intestinal flora to scurvy of guinea pigs and of infants.

Uhlenhuth, E.

1308. Does the thymus gland of mammals when given as food to Amphibians exert any specific influence upon the organism?

Unger, Lester J.

1336. See **Hess, Alfred F.**

1371. See **Hess Alfred F.**

Walsh, L. S. N.

1370. [with **Leo Loeb.**] A note on the healing in and migration of foreign bodies in the animal body.

Wardell, Emma L.

1283. See **Kast, Ludwig.**

1343. See **Kast, Ludwig.**

Watanabe, C. K.

1372. [with **V. C. Myers.**] A delicate method of determining invert activity.

1373. The phosphate and calcium content of serum in the condition of guanidine tetany.

Weiss, Charles.

1342. [with **John A. Kolmer.**] A skin reaction to pneumotoxin.

West, C. J.

1305. See **Levene, P. A.**

Wiggers, C. J.

1306. Fat emboli and shock.

Winslow, C.-E. A.

1326. [with **I. S. Falk.**] Studies on salt action. 1. Effect of calcium and sodium salts upon the viability of the colon bacillus in water.

1359. [with **David Greenberg.**] The effect of the respiration of putrid gases upon the growth of guinea pigs.

1365. [with **I. S. Falk.**] Studies on salt action II. Effect of transfer from stronger to weaker salt solutions upon the viability of bacteria.

Wollstein, Martha.

1321. [with **S. J. Meltzer.**] Chemical pneumonia.

EXECUTIVE PROCEEDINGS.

Main Society.

Eighty-fifth Meeting.

Cornell University Medical College, October 17, 1917. President Gies in the chair.

Members present: Auer, Benedict, Gies, Githens, Jackson, Kleiner, Lusk, Meltzer, Morgan, Myers, V. C., Ringer, Wiggers.

Members elected: Walter C. Alvarez, G. D. Barnett, George W. Corner, Harold K. Faber, F. L. Gates, Maurice H. Givens, C. Lundsgaard.

Eighty-sixth Meeting.

New York Post Graduate Medical School, November 21, 1917. President Gies in the chair.

Members present: Auer, Falk, Funk, Gies, Githens, Goldfarb, Jackson, Janney, Kleiner, Lambert, Levine, Meyer, G. M., Moore, Myers, V. C.

Members elected: David I. Macht, George B. Roth.

Eighty-seventh Meeting.

Rockefeller Institute for Medical Research, December 19, 1917. President Gies in the chair.

Members present: Amoss, Auer, Austin, Cole, Edwards, Falk, Flexner, Gies, Githens, Goldfarb, Jackson, Kleiner, Lambert, Levene, Loeb, J., Lusk, Meyer, A. L., Meyer, G. M., Pike, Ringer, Terry, Uhlenhuth, Van Slyke, West, Wiggers.

Eighty-eighth Meeting.

College of Physicians and Surgeons, January 16, 1918. President Gies in the chair.

Members present: Auer, Coleman, Edwards, Falk, Gies, Jackson, Kleiner, Lambert, Lieb, Meltzer, Meyer, A. L., Myers, Pike, Riddle, Sherman, Uhlenhuth, Wadsworth, Wiggers, Wollstein.

Eighty-ninth Meeting. (Fifteenth Annual Meeting.)

College of the City of New York, February 20, 1918. President Gies in the chair.

Members present: Auer, Benedict, Gies, Githens, Givens, Goldfarb, Hess, Jackson, Janney, Kober, Macht, Meltzer, Mendel, Meyer, A. L., Peirce, Sherman, Wadsworth, Wiggers, Winslow.

Member elected: A. R. Rose.

The meeting was held at 5.00 P. M., and was followed by a dinner at 7.15 P. M. Election of officers occurred for the ensuing year after the dinner and resulted as follows:

President, William J. Gies; Vice-President, John Auer; Secretary-Treasurer, Holmes C. Jackson; additional members of the Council, George B. Wallace, Henry C. Sherman.

Ninetieth Meeting.

Presbyterian Hospital, March 20, 1918. President Gies in the chair.

Members present: Coleman, Epstein, Gies, Githens, Goldfarb, Hess, Jackson, Kleiner, Lambert, Lieb, Loeb, J., Meltzer, Northrop, Olmstead, Salant, Sherman, Uhlenhuth.

Members elected: D. E. Jackson, Arthur Knudson, C. P. Sherwin, Arthur W. Thomas.

Ninety-first Meeting.

University and Bellevue Hospital Medical College, April 17, 1918. President Gies in the chair.

Members present: Auer, Erdmann, Gies, Githens, Givens, Hess, Hoskins, E. R., Kast, Lambert, Myers, V. C., Jackson, Sherman, Uhlenhuth.

Members elected: A. K. Balls, W. W. Palmer.

Ninety-second Meeting.

Columbia University, May 15, 1918. President Gies in the chair.

Members present: Gies, Githens, Goldfarb, Hess, Jackson, Lambert, Loeb, J., Meltzer, Meyer, A. L., Morgan, Myers, V. C., Peirce, Sherman, Stockard, Uhlenhuth, Wilson, Winslow.

Members elected: B. M. Allen, J. A. Killian.

PACIFIC COAST BRANCH.

Eighteenth Meeting.

San Francisco, California, October 3, 1917.

Members present: Addis, Burnett, Cooke, Dickson, Hewlett, Hooper, Hurwitz, Walker, Whipple.

Nineteenth Meeting.

San Francisco, California, February 6, 1918.

Members present: Addis, Alvarez, Burnett, Dickson, Evans, Hooper, Whipple..

Twentieth Meeting.

San Francisco, California, April, 8, 1918.

Members present: Alvarez, Burnett, Corner, Ophüls, Schmidt, Walker, Whipple.

REGISTER OF NAMES AND ADDRESSES OF THE MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

ABBOTT, ALEXANDER C.....University of Pennsylvania.
ABEL, JOHN J.....Johns Hopkins University.
ADAMI, J. GEORGE.....McGill University, Montreal.
ADDIS, THOMAS.....Leland Stanford University, San Francisco.
ADLER, HERMAN M.....Juvenile Psychopathic Institute, Chicago.
ALLEN, A. REGINALD.....University of Pennsylvania.
ALLEN, BENNET M.....University of Kansas.
ALSBERG, CARL.....U. S. Department of Agriculture, Washington, D. C.
ALVAREZ, WALTER C.....University of California.
AMOSS, HAROLD L.....Rockefeller Institute for Medical Research.
ANDERSON, JOHN F.....New Brunswick, N. J.
ATKINSON, JAMES P.....Department of Health, New York City.
AUER, JOHN.....Rockefeller Institute for Medical Research.
AUSTIN, J. H.....University of Pennsylvania.

BAEHR, GEORGE.....Mt. Sinai Hospital, N. Y. City.
BAILEY, CHARLES H.....Tufts College.
BAILEY, HAROLD.....Cornell University Medical College.
BAITSELL, G. A.....Yale University.
BALLS, A. K.....Columbia University.
BANTA, A. M.....Carnegie Institution, Station for Experimental Evolution, Cold Spring Harbor, Long Island, N. Y.
BANZHAF, EDWIN J.....Department of Health, New York City.
BARBER, W. H.....New York University.
BARBOUR, HENRY G.....Yale University.
BARDEEN, CHARLES R.....University of Wisconsin.
BARNETT, GEORGE D.....Leland Stanford Jr., University.
BAUMANN, LOUIS.....University of Iowa.
BENEDICT, STANLEY R.....Cornell University Medical College.
BERG, WILLIAM N.....U. S. Department of Agriculture, Washington, D. C.
BERGEIM, O.....Jefferson Medical College, Philadelphia, Pa.
BERGEY, DAVID H.....University of Pennsylvania.
BEUTNER, REINHARD.....Germany.
BIRCHARD, F. J.....Dominion Laboratory, Winnipeg, Man., Canada.
BRONFENBRENNER, JACOB.....Harvard Medical School.
BROOKS, HARLOW.....New York University.
BROWN, WADE H.....Rockefeller Institute for Medical Research.
BROWNE, WILLIAM W.....College of City of New York.
BULL, C. G.....Rockefeller Institute for Medical Research.
BUNTING, C. H.....University of Wisconsin.
BURNETT, T. C.....University of California.

BURROWS, M. J. ..... Washington University Medical School.
BURTON-OPITZ, RUSSELL. ..... Columbia University.
BUTTERFIELD, E. E. ..... 135 E. 34th Street, N. Y. City.

CALKINS, GARY N. ..... Columbia University.
CANNON, WALTER B. ..... Harvard University.
CARLSON, A. J. ..... University of Chicago.
CARREL, ALEXIS ..... Rockefeller Institute for Medical Research.
CAULFEILD, A. H. ..... University of Toronto, Toronto, Can.
CECIL, R. L. ..... Presbyterian Hospital, Columbia University.
CHIDESTER, F. E. ..... Rutgers College, New Brunswick, N. J.
CHITTENDEN, R. H. ..... Yale University.
CHURCHMAN, J. W. ..... Yale University.
CLARK, P. F. ..... University of Wisconsin.
CLOWES, G. H. A. ..... Gratwick Laboratory, Buffalo, N. Y.
COCA, A. F. ..... New York Hospital.
COHN, ALFRED E. ..... Rockefeller Institute for Medical Research.
COLE, L. J. ..... University of Wisconsin.
COLE, RUFUS I. ..... Rockefeller Institute for Medical Research.
COLEMAN, W. ..... New York University.
COLLINS, KATHARINE R. ..... 8803 Euclid Avenue, Cleveland, Ohio.
CONKLIN, EDWIN G. ..... Princeton University.
COOKE, J. V. ..... University of California.
CORNER, GEORGE W. ..... University of California.
COUNCILMAN, WILLIAM T. ..... Harvard University.
CRAMPTON, C. WARD ..... Department of Education, New York City.
CRAWFORD, ALBERT C. ..... Leland Stanford University.
CRILE, GEORGE W. ..... Western Reserve University, Cleveland.
CUSHING, HARVEY ..... Harvard University.

DAKIN, H. D. ..... 819 Madison Avenue, New York City.
DANDY, WALTER E. ..... Johns Hopkins University.
DAVENPORT, CHARLES B. Carnegie Institution, Station for Experimental Evolution, Cold Spring Harbor, Long Island, N. Y.
DICKSON, E. C. ..... Stanford University Medical School.
DOCHEZ, A. R. ..... Rockefeller Institute for Medical Research.
DONALDSON, H. H. ..... Wistar Institute of Anatomy, Philadelphia.
DRAPER, GEORGE ..... Presbyterian Hospital, Columbia University.
DRAPER, J. W. ..... New York University.
DRESBACH, M. ..... Cornell University.
DUBOIS, E. F. ..... Cornell University Medical College.
DUNHAM, EDWARD K. ..... New York University.
DUVAL, CHARLES W. ..... Tulane University.

EDDY, WALTER H. ..... High School of Commerce Annex, New York City.
EDMUNDS, C. W. ..... University of Michigan.
EDSALL, DAVID L. ..... Harvard University.
EDWARDS, D. J. ..... Cornell University Medical College.
EISENBREY, A. B. ..... Western Reserve University.

EGGLESTON, CARY.....Cornell University Medical College.
ELSBERG, CHARLES A.....Mount Sinai Hospital.
ELSER, WILLIAM J.....Cornell University Medical College.
EMERSON, HAVEN.....Columbia University.
EPSTEIN, ALBERT A.....Mt. Sinai Hospital, N. Y.
ERDMANN, RHODA.....Yale University.
ERLANGER, JOSEPH.....Washington University, St. Louis.
EVANS, H. M.....University of California.
EWING, E. M.....Silver City, New Mexico.
EWING, JAMES.....Cornell University Medical College.
EYSTER, J. A. E.....University of Wisconsin.

FABER, HAROLD K.....Leland Stanford Jr. University.
FAHR, GEORGE.....Montefiore Home and Hospital, N. Y. City.
FALK, K. G...Harriman Research Laboratory, Roosevelt Hospital, New York City.
FAMULENER, L. W.....St. Luke's Hospital, New York City.
FIELD, CYRUS W.....24 E. 48th Street, New York City.
FINE, M. S.....Calco Chemical Co., Bound Brook, N. J.
FISCHER, MARTIN H.....General Hospital, Cincinnati.
FITZGERALD, J. G.....University of Toronto, Toronto, Canada.
FITZPATRICK, C. B.....Department of Health, New York City.
FLEXNER, SIMON.....Rockefeller Institute for Medical Research.
FLOURNOY, THOMAS.....Mercy Hospital, Pittsfield, Mass.
FOLIN, OTTO.....Harvard University.
FORD, WILLIAM W.....Johns Hopkins University.
FOSTER, NELLIS B.....University of Michigan.
FROST, W. H.....Ohio River Investigation, Cincinnati, Ohio.
FUNK, CASIMIR.....215 Manhattan Ave., N. Y. City.

GAGER, C. STUART.....Brooklyn Botanic Garden.
GATES, F. L.....Rockefeller Institute.
GAY, FREDERICK P.....University of California.
GAYLORD, H. R.....Gratwick Laboratory, Buffalo, N. Y.
GETTLER, A. O.....New York University.
GIBSON, ROBERT B.....Philippine Medical School, Manila, P. I.
GIES, WILLIAM J.....Columbia University.
GITHENS, T. S.....Rockefeller Institute for Medical Research.
GIVENS, MAURICE H.....University of Rochester.
GLASER, OTTO C.....University of Michigan.
GOLDFARB, A. J.....College of the City of New York.
GORTNER, R. A.....University of Minnesota.
GREENWALD, I.....Harriman Research Laboratory, Roosevelt Hospital, N. Y. City.
GUENTHER, A. E.....University of Nebraska, Lincoln, Nebraska.
GUTHRIE, C. C.....University of Pittsburgh.

HALE, WORTH.....Harvard University.
HALSTED, WILLIAM S.....Johns Hopkins University.
HANZLIK, P. J.....Western Reserve Medical School, Cleveland, Ohio.
HARRIS, ISAAC F.....New Brunswick, N. J.
HARRISON, ROSS G.....Yale University.

HARTWELL, J. A. ..... Cornell University Medical College.
HATCHER, ROBERT A. ..... Cornell University Medical College.
HATAI, SHINKISHI ..... Wistar Institute of Anatomy.
HAWK, PHILIP B. ..... Jefferson Medical College, Philadelphia, Pa.
HESS, ALFRED F. ..... Department of Health, New York City.
HEWLETT, A. W. ..... Stanford Medical School.
HIRSCHFELDER, A. D. ..... University of Minnesota.
HODGE, C. F. ..... University of Oregon.
HOLMAN, W. L. ..... University of Pittsburgh.
HOLMES, S. J. ..... University of California.
HOOKER, DAVENPORT ..... Yale University.
HOOPER, C. H. ..... University of California.
HOPKINS, J. GARDNER ..... Columbia University.
HOSKINS, E. R. ..... New York University.
HOSKINS, R. G. ..... Northwestern University.
HOWE, P. E. ..... Rockefeller Institute, Princeton, N. J.
HOWELL, WILLIAM H. ..... Johns Hopkins University.
HOWLAND, JOHN ..... Johns Hopkins University.
HUBER, G. CARL ..... University of Michigan.
HUNT, REID ..... Harvard University.
HUNTER, ANDREW ..... University of Toronto.
HURWITZ, SAMUEL H. ..... University of California.

JACKSON, D. E. ..... Washington University Medical School.
JACKSON, HOLMES C. ..... New York University.
JACOBS, WALTER A. ..... Rockefeller Institute for Medical Research.
JANEWAY, H. H. ..... Memorial Hospital, N. Y. City.
JANNEY, NELSON W. ..... Montefiore Home and Hospital, N. Y. City.
JENNINGS, H. S. ..... Johns Hopkins University.
JOBLING, JAMES W. ..... Vanderbilt University, Nashville.
JONES, F. S. ..... Rockefeller Institute, Princeton, N. J.
JONES, WALTER ..... Johns Hopkins University.
JORDAN, H. E. ..... University of Virginia.
JOSEPH, DON R. ..... St. Louis University Medical School.

KAHN, MAX ..... Beth Israel Hospital, N. Y. City.
KAHN, R. L. ..... Department Laboratory, Atlanta, Georgia.
KARSNER, H. T. ..... Western Reserve Medical College.
KAST, LUDWIG ..... New York Post-Gratuate Medical School.
KELLOGG, V. L. ..... Stanford University.
KILLIAN, JOHN A. ..... New York Post-Graduate Medical School.
KINSELLA, RALPH A. ..... Presbyterian Hospital, N. Y. City.
KIRKBRIDE, MARY B. ..... State Hygienic Laboratory, Albany, N. Y.
KLEINER, I. S. ..... Rockefeller Institute for Medical Research.
KLINE, B. S. ..... Montefiore Home and Hospital, N. Y. City.
KLOTZ, OSKAR ..... University of Pittsburgh.
KNUDSON, ARTHUR ..... Albany Medical College.
KOBER, P. A. ..... State Department of Health, Albany, N. Y.
KOCHER, R. A. ..... University of California.
KOLMER, J. A. ..... University of Pennsylvania.
KRUMBHAAR, E. B. ..... University of Pennsylvania.

LAMAR, RICHARD V. ..... University of Georgia.
LAMBERT, R. A. ..... Columbia University.
LAURENS, HENRY ..... Yale University.
LEATHES, J. B. ..... Sheffield University.
LEE, FREDERIC S. ..... Columbia University.
LEVENE, P. A. ..... Rockefeller Institute for Medical Research.
LEVIN, ISAAC ..... Columbia University.
LEWIS, H. B. ..... University of Illinois.
LEWIS, PAUL A. ..... Phipps Institute, Philadelphia.
LIEB, C. C. ..... Columbia University.
LILLIE, FRANK R. ..... University of Chicago.
LILLIE, RALPH S. ..... Clark University.
LOEB, JACQUES ..... Rockefeller Institute for Medical Research.
LOEB, LEO ..... Washington University, St. Louis.
LOEVENHART, ARTHUR S. ..... University of Wisconsin.
LOMBARD, WARREN P. ..... University of Michigan.
LONGCOPE, W. T. ..... Presbyterian Hospital, Columbia University.
LUCAS, W. P. ..... University of California.
LUNDSGUARD, CHRISTIAN ..... University of Copenhagen, Denmark.
LUSK, GRAHAM ..... Cornell University Medical College.
LYLE, W. L. ..... Harriman Research Laboratory, Roosevelt Hospital, N. Y. C.
LYON, E. P. ..... University of Minnesota.

MACALLUM, A. B. ..... University of Toronto.
MACCALLUM, W. G. ..... Johns Hopkins University.
MACDOUGAL, D. T. ..... Desert Laboratory, Tucson, Arizona.
MACLEOD, J. J. R. ..... Western Reserve University, Cleveland.
MACNEAL, WARD J. ..... New York Post-Graduate Medical School.
MACNIDER, W. DEB. ..... University of North Carolina.
MCCRUDDEN, F. M. ..... Robert Brigham Hospital, Boston, Mass.
MCLEAN, FRANKLIN C. ..... Peking Union Medical College.
MCMEANS, J. W. ..... University of Pittsburgh.
MACHT, DAVID I. ..... Johns Hopkins University.
MANDEL, ARTHUR R. ..... New York University.
MANDEL, JOHN A. ..... New York University.
MANWARING, W. H. ..... Leland Stanford University.
MARINE, DAVID ..... Western Reserve University, Cleveland.
MAXWELL, S. S. ..... University of California.
MAYER, ALFRED G. ..... Carnegie Institution, Washington, D. C.
MEIGS, EDWARD B. ..... Dairy Division Experiment Station, Beltsville, Md.
MELTZER, S. J. ..... Rockefeller Institute for Medical Research.
MENDEL, LAFAYETTE B. ..... Yale University.
MEYER, ADOLPH ..... Johns Hopkins University.
MEYER, ARTHUR L. ..... Rockefeller Institute for Medical Research.
MEYER, GUSTAVE M. ..... Rockefeller Institute for Medical Research.
MEYER, K. F. ..... University of California.
MOHLER, J. R. ..... Bureau of Animal Industry, Washington, D. C.
MOORE, A. R. ..... Rutgers College.
MORGAN, THOMAS H. ..... Columbia University.
MORSE, WITHROW ..... Morris Institute, Chicago.

MOSENTHAL, HERMAN O.....Johns Hopkins Hospital.
MURLIN, JOHN R.....University of Rochester.
MURPHY, JAMES B.....Rockefeller Institute for Medical Research.
MYERS, V. C.....New York Post-Graduate Medical School.

NILES, WALTER L.....56 E. 77th Street, New York City.
NOBLE, W. C.....New York University.
NOGUCHI, HIDEYO.....Rockefeller Institute for Medical Research.
NORRIS, CHARLES.....Medical Director, New York City.
NORTHROP, JOHN H.....Rockefeller Institute for Medical Research.
NOVY, FREDERICK G.....University of Michigan.

OERTEL, HORST.....Royal Victoria Hospital, Montreal.
OLMSTEAD, MIRIAM P.....Presbyterian Hospital, New York City.
OPHÜLS, WILLIAM.....Leland Stanford University.
OPIE, EUGENE L.....Washington University, St. Louis.
OPPENHEIMER, B. S.....Columbia University.
OSBORNE, THOMAS B.....Connecticut Agricultural Experiment Station, New Haven, Conn.
OSTERHOUT, W. J.....Harvard University.
OTTENBERG, R.....Mount Sinai Hospital.

PALMER, WALTER W.....Presbyterian Hospital.
PAPPENHEIMER, ALVIN M.....Columbia University.
PARK, E. A.....Johns Hopkins University.
PARK, WILLIAM H.....New York University.
PARKER, GEORGE H.....Harvard University.
PEABODY, FRANCIS W.....Peter Bent Brigham Hospital, Boston, Mass.
PEARCE, LOUISE.....Rockefeller Institute for Medical Research.
PEARCE, RICHARD M.....University of Pennsylvania.
PEARL, RAYMOND.....Maine Agricultural Experiment Station, Orono, Maine.
PEIRCE, GEORGE.....38 Godfrey Road, Montclair, N. J.
PEMBERTON, RALPH.....Presbyterian Hospital, Philadelphia, Pa.
PETERSEN, W. F.....Vanderbilt University.
PEPPER, O. H. PERRY.....University of Pennsylvania.
PFAFF, F.....Harvard University.
PFEIFFER, J. A. F.....Government Hospital for Insane, Washington, D. C.
PIKE, F. H.....Columbia University.
PLOTZ, HARRY.....Mt. Sinai Hospital, N. Y. City.
PORTER, WILLIAM T.....Harvard University.
PRATT, JOSEPH H.....Harvard University.
PRINCE, A. L.....Yale University.
RAVENEL, MAZŸCK P.....University of Missouri.
REICHERT, EDWARD T.....University of Pennsylvania.
RETTGER, LEO F.....Yale University.
RICHARDS, ALFRED N.....University of Pennsylvania.
RIDDLE, O.....Station for Experimental Evolution, Cold Spring Harbor, N. Y.
RINGER, A. I.....141 W. 78th Street, N. Y. City.
ROBERTSON, T. BRAILSFORD.....University of Toronto.
ROBINSON, G. CANBY.....Washington University, St. Louis.

ROSE, ANTON R.....New York Post-Graduate Medical School.
ROSENAU, MILTON J.....Harvard University.
ROSENBLOOM, JACOB.....Western Pennsylvania Hospital, Pittsburgh, Pa.
ROTH, GEORGE B.....University of Kansas.
ROTHSCHILD, M. A.....Mt. Sinai Hospital, N. Y. City.
ROUS, PEYTON.....Rockefeller Institute for Medical Research.

SALANT, WILLIAM.....U. S. Department of Agriculture, Washington, D. C.
SCHULTZ, F. W.....University of Minnesota.
SCHMIDT, CARL L. A.....University of California.
SCHULTZ, W. H.....West Virginia University.
SCHWYZER, FRITZ.....Kastanienbaum, near Luzern, Switzerland.
SCOTT, E. L.....Columbia University.
SCOTT, G. G.....College of the City of New York.
SENIOR, H. D.....New York University.
SHAFFER, PHILIP A.....Washington University, St. Louis.
SHAKLEE, A. O.....Fordham University.
SHERMAN, HENRY C.....Columbia University.
SHERWIN, CARL P.....Fordham University.
SILER, J. F..Department Laboratory, Southern Department, Fort Sam Houston. Texas.
SIMON, CHARLES E.....University of Maryland.
SIMPSON, SUTHERLAND.....Cornell University, Ithaca, N. Y.
SITTENFIELD, M. J.....Columbia University.
SMITH, THEOBALD.....Rockefeller Institute, Princeton, N.J.
SOLLMAN, TORALD.....Western Reserve University, Cleveland.
SOUTHARD, E. E.....70 Francis Avenue, Cambridge, Mass.
SPAETH, REYNOLD ALBRECHT.....Yale University.
STEWART, GEORGE N.....Western Reserve University, Cleveland.
STILES, PERCY G.....Harvard University.
STOCKARD, CHARLES R.....Cornell University Medical College.
STOOKEY, LYMAN B.....University of Southern California, Los Angeles.
STOREY, THOMAS A.....College of the City of New York.
STRONG, RICHARD P.....Harvard University.
STROUSE, SOLOMON.....104 S. Michigan Ave., Chicago, Illinois.
SWAIN, R. E.....Stanford University, California.
SWEET, J. EDWIN.....University of Pennsylvania.
SWIFT, H. F.....Columbia University.
SYMMERS, DOUGLAS.....New York University.

TASHIRO, SHIRO.....University of Chicago.
TAYLOR, ALONZO E.....University of Pennsylvania.
TAYLOR, R. M.....New York Post-Graduate Medical School.
TEAGUE, OSCAR.....Quarantine Laboratory, Rosebank, N. Y.
TEN BROECK, CARL.....Rockefeller Institute, Princeton, N. J.
TERRY, B. T.....Vanderbilt University.
THOMAS, ARTHUR W.....Columbia University.
THRO, W. C.....Cornell University Medical College.
TODD, JOHN L.....McGill University, Montreal.
TORREY, JOHN C.....Cornell University Medical College.
TYZZER, E. E.....Harvard University.

UHLENHUTH, EDWARD.....Rockefeller Institute for Medical Research.
UNDERHILL, FRANK P.....Yale University.

VAN SLYKE, DONALD D.....Rockefeller Institute for Medical Research.

WADSWORTH, AUGUSTUS B.....State Department of Health, Albany, N. Y.
WALLACE, GEORGE B.....New York University.
WALKER, E. L.....University of California.
WARTHIN, ALFRED S.....University of Michigan.
WASTENEYS, H.....University of California.
WATANABE, C. K.....1631 Post Street, San Francisco, Calif.
WELCH, WILLIAM H.....Johns Hopkins University.
WELKER, WILLIAM H.....University of Illinois.
WELLER, CARL VERNON.....University of Michigan.
WELLS, H. GIDEON.....University of Chicago.
WEST, C. J.....3315 Wisconsin Ave., N. W., Washington, D. C.
WHIPPLE, G. H.....University of California.
WHITE, BENJAMIN.....Otisville, N. Y.
WHITE, O. E.....Brooklyn Botanic Garden, Brooklyn, N. Y.
WIGGERS, C. J.....Cornell University Medical College.
WILLIAMS, ANNA W.....Department of Health, New York City.
WILLIAMS, H. B.....Columbia University.
WILLIAMS, HERBERT U.....University of Buffalo.
WILSON, EDMUND B.....Columbia University.
WINSLOW, C.-E. A.....Yale University.
WOLBACH, S. BURT.....Harvard University.
WOLF, CHARLES G. L.....Cambridge, England.
WOLLSTEIN, MARTHA.....Rockefeller Institude for Medical Research.
WOOD, FRANCIS C.....Columbia University.
WOODRUFF, LORANDE LOSS.....Yale University.

YATSU, NAOHIDÉ.....University of Japan.
YERKES, ROBERT M.....Harvard University.

ZINGHER, A.....Department of Health, New York City.
ZINSSER, HANS.....Columbia University.

Total number of members at the close of the academic year, 1917–18: 355.

OFFICERS.

1903–1918.

	1903–'04	1904–'05	1905–'06	1906–'07	1907–'08	1908–'09
President	Meltzer	Meltzer	Wilson	Flexner	Flexner	Lee
Vice-President	Park	Ewing	Dunham	Dunham	Morgan	Morgan
Librarian	Lusk	Lusk	Lusk	——	——	——
Treasurer	Calkins	Calkins	Calkins	Calkins	Calkins	Lusk
Secretary	Gies	Gies	Gies	Gies	Gies	Gies

	1909–'10	1910–'11	1911–'12	1912–'13	1913–'14	1914–'15
President	Lee	Morgan	Morgan	Ewing	Ewing	Lusk
Vice-Presidient	Gies	Gies	Levene	Levene	Field	Gies
Treasurer	Lusk	Lusk	Lusk	Norris	Norris	Murlin
Secretary	Opie	Opie	Wallace	Wallace	Jackson	Jackson

	1915–'16	1916–'17	1917–'18	1918–'19
President	Lusk	Jacques Loeb	Gies	Gies
Vice-President	Calkins	Gies	Auer	Auer
Sec'y-Treas	Jackson	Jackson	Jackson	Jackson
Additional members of Council[1]	Gies	Auer	Dubois	Wallace
	Auer	Dubois	Wallace	Sherman

[1] The Past Presidents are also members.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York.)

College of the City of New York.—W. W. Browne, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—A. K. Balls, Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, George Draper, Haven Emerson, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, Arthur W. Thomas, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, E. F. DuBois, D. J. Edwards, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, Charles R. Stockard, W. C. Thro, John C. Torrey, C. J. Wiggers.

Fordham University School of Medicine.—A. O. Shaklee, Carl P. Sherwin.

Hospitals, Bellevue.—Charles Norris. *Beth Israel.*—Max Kahn. *Memorial Hospital.*—H. H. Janeway. *Montefiore Home.*—George Fahr, Nelson W. Janney, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead, Walter H. Palmer. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Post-Graduate Medical School.—Ludwig Kast, John A. Killian, W. J. MacNeal, V. C. Myers, Anton R. Rose, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, F. L. Gates, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 E. 34th St., N. Y. City.—E. E. Butterfield.

819 Madison Avenue, N. Y. City.—H. D. Dakin.

High School of Commerce Annex, 197 E. Broadway, N. Y. City.—Walter H. Eddy.

24 East 48th St., N. Y. City.—Cyrus W. Field.

215 Manhattan Ave., N. Y. City.—Casimir Funk.

56 East 77th St., N. Y. City.—Walter L. Niles.
141 W. 78th St., N. Y. City.—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (New Haven).—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs.

Carnegie Institution of Washington. (Station for Experimental Evolution, Cold Spring Harbor, N. Y.).—A. M. Banta, Charles B. Davenport, O. Riddle. *(Desert Laboratory, Tucson, Ariz.).*—D. T. MacDougal. *(Tortugas, Florida).*—Alfred G. Mayer.

State Boards of Health. New York (Albany).—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (Cincinnati, Ohio).—Martin H. Fischer. *Government Hospital for Insane (Washington, D. C.).*—J. A. F. Pfeiffer. *Mercy (Pittsfield, Mass.).*—Thomas Flournoy. *Peter Bent Brigham (Boston).*—Francis W. Peabody. *Presbyterian (Phila.).*—Ralph Pemberton. *Robert Brigham (Boston).*—F. M. McCrudden. *Royal Victoria (Montreal).*—Horst Oertel. *Western Pennsylvania (Pittsburgh).*—Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Juvenile Psychopathic (Chicago).*—Herman M. Adler. *Morris (Chicago).*—Withrow Morse. *Phipps (Philadelphia).*—Paul A. Lewis. *Rockefeller (Princeton).*—F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Department Laboratory (Atlanta, Ga.).*—R. L. Kahn. *Medical Corps (Fort San Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—Walter C. Alvarez, T. C. Burnett, J. V. Cooke, George W. Corner, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, C. K. Watanabe, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Copenhagen.*—Christen Lundsgaard. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V. Lamar. *Harvard.*—Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, W. J. V. Osterhout, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidí Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, H. S. Jennings, Walter Jones, David L. Macht, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Kansas.*—Bennet M. Allen, George B. Roth. *Leland Stanford.*—Thomas Addis, George D. Barnett, A. C. Crawford, E. C. Dickson, Harold K. Faber, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P.

Lyon, F. W. Schultz. *Missouri.*—Mazyck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rochester.*—Maurice H. Givens, John R. Murlin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum, T. Brailsford Robertson. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Union University (Albany Medical College).*—Arthur Knudson. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen, B. T. Terry. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. T. Burrows, Joseph Erlanger, D. E. Jackson, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Rhoda Erdmann, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.

Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.

Cleveland, Ohio, 8803 Euclid Ave.—Katharine R. Collins.

Kastanienbaum, Switzerland.—Fritz Schwyzer.

Calco Chemical Co., Bound Brook, N. J.—M. S. Fine.

New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.

Otisville, N. Y.—B. White.

Rosebank, N. Y.—Oscar Teague.

Silver City, New Mexico.—E. M. Ewing.

Upper Montclair, N. J.—George Peirce.

Washington, D. C., 3315 Wisconsin Ave.—C. J. West.

Winnipeg, Canada.—F. J. Birchard.

Germany.—Beutner, Reinhard.

INDEX

OF THE

SCIENTIFIC PROCEEDINGS

[THE NUMERALS IN THE INDEX CORRESPOND WITH THE NUMERALS IN PARENTHESIS ABOVE THE TITLES OF THE ABSTRACTS. PAGES ARE NOT INDICATED.]

Acid, development of, in nervous tissue, 1297.
Adrenalin, causing glycemia and glycosuria, 1286.
Alkali, therapy effect of, on diastase and sugar of blood, 1296.
Alkaloids, effect of, on bacteria and protozoa of intestine, 1360.
Amylase action of pancreatic preparations, 1318.
Analgesia, after exposure to dimethylsulphate, 1350.
Antipyretics, influence of, on coördination test, 1323; on field of vision, 1313.
Antipoliomyelitic serum, experiments with Rosenow's, 1311.
Antiscorbutic power, effect of treatment of cabbage on, 1361.
Antiscorbutic, intravenous injection of, 1371.
Antiseptics, intravenous injection of an, 1371.
Antiseptics, effect of, on bacteria and protozoa in intestine, 1360; solvent action of on necrotic tissue, 1303.
Apomorphine, absorption of, 1302.
Ataxia in pigeons, 1320.

Bacteria, effect of antiseptics on, in intestine, 1360; effect of salt solutions upon viability of, 1365.
Biocolloids, swelling of, effect of temperature on, 1314.
Bladder, absorptive power for drugs of, 1312.
Blood, tolerance, test for sugar in, 1294; urea content of, 1343.

Cabbage, effect of treatment of, on antiscorbutic power, 1361.
Calcium, content of dried vegetables, 1347; metabolism, effect of acid and dietary factors on, 1325; salts, effect of, on viability of colon bacillus, 1326; in blood, in abnormal conditions, 1291.
Caries, dental, 1287.
Carnosine, synthesis of, 1375.
Castration, effect of, on cockerels, 1285.
Catalase accelerator in liver, 1334.
Cell, division, effect of, by temperature, 1357.
Cells, embryonic, toxicity of dipeptids upon, 1369.
Cerebellum, effect upon, by nitrobenzene, 1367; lesions in dogs, 1317.
Cerebral lesions, in dogs, 1317.
Cephalin, relation to hydrolecithin, 1305.
Cholesterol in blood, 1283.
Coagulation of blood, influence of hypochlorite solution on, 1362.
Conductance, intragastric, 1290.
Creatine, oxidation product of, 1374.
Cytidin phosphoric acid, 1299.

Dextrose, criticism of picrate method for, 1333.
Diabetes, morphin test in, 1339; protein sparing by glucose in, 1327.
Diastase of blood, influence of anesthesia and alkali on, 1296.
Dimethylsulphate, analgesia after exposure to, 1350; pulmonary edema after inhalation of, 1351.
Dipeptids, toxicity of, upon embryonic cells, 1369.
Duodenal contents, fractional analysis of, 1368.

Edema, pulmonary, after inhalation of dimethylsulphate, 1351.

Fat, relation to shock, 1306.
Fertilization, self incompatibilities in, 1316.
Foreign bodies, healing in and migration of, in body, 1370.

Gall, crown, influence of X-rays on, 1300.
Gases, putrid, effect of, upon growth of guinea pigs, 1359.

Gastric contents, fractional analysis of, 1368.
Gelatin solution, influence of netural salts upon, 1364.
Glycin, two forms of, 1301.
Growth, effect of respiration of putrid gases upon, 1359; influence of temperature upon, 1314; method of measurement of, 1298.
Growths, transplantable production of, 1344.
Guanidine tetany, salt content of serum in, 1373.

Hydrolecithin, relation to cephalin, 1305.
Hyperglycemia morphin, test for pancreas, 1284.
Hypochlorite solution, Dakin's, action of, on blood clotting, 1362; preparation of Dakin's, 1310.

Immunity, persistence of, in toxin-antitoxin injections, 1346.
Immunization of monkeys against infantile paralysis, 1345.
Inbreeding, effect of, on tumor rate in mice, 1330.
Incompatibilities, self, in fertilization, 1316.
Infantile paralysis, monkey immunization against, 1345.
Intestinal flora, relation of scurvy to, 1332.
Invert activity, method of determination of, 1372.

Lipemia in diabetes mellitus, 1309.
Liver, catalase accelerator in, 1334; sulpho-conjugation as test of function of, 1331.

Magnesium metabolism, effect of acid and dietary factors on, 1325.
Magnesium sulphate, effect upon tetanus, 1307.
Morphin, absorption of, 1302; test in diabetes, 1339.
Muscle, smooth, effect of opium alkaloids on, 1324.

Necrotic tissue, solvent action of, by antiseptics, 1303.
Nephritis, renal functions in, 1338; tartrate, 1288.
Nerve, development of acid in, 1297.
Nitrobenzene, action of, upon cerebellum, 1367.
Nitrogen content of dried vegetables, 1347; method for minute quantities of, 1340.
Nutrition in animal tissues, 1329.
Opiates, influence of, on field of vision, 1313.
Opium, structure of alkaloids of, and effect of on smooth muscle, 1324.

Pancreas, effect of adrenalin on glycemia and glycosuria, 1286.
Paratyphoid A and B, differentiation of typhoid and, 1304.
Picrate method for dextrose, criticism of, 1333.
Pneumococci, in mouth before operation, 1337.
Pneumonia, chemical, 1321.
Polarity, in regeneration, chemical basis of, 1315.
Protease action of pancreatic preparations, 1318.
Protein sparing by glucose in diabetes, 1327.
Protozoa, effect of antiseptics on, in intestine, 1360.

Reflex thresholds in shock, 1322.
Respiration, afferent impulses in control of, 1319; of putrid gases on growth, 1359.
Respirations, minimum number of, for life, 1363.
Roughage, effect of, on experimental scurvy, 1358.

Saccharose, inversion and determination of, 1295.
Salt solutions, effect upon viability by transfer from stronger to weaker, 1365.
Salts, neutral, influence of, upon gelatin solution, 1364.
Scurvy, 1336; relation of diet and roughage to, 1358; in guinea pigs production of, 1348; relation of intestinal flora to, 1332.
Shock, experimental, 1328; fat emboli and, 1306; reflex thresholds in, 1322.
Sodium salts, effect of, on viability of colon bacillus, 1326.
Sugar, blood, tolerance test for, 1294; influence of anesthesia and alkali on, 1296; rate of dialysis of, 1335.
Sulpho-conjugation as test of hepatic function, 1331.

Tartrate nephritis, renal activity in, 1289.
Temperature, effect of upon cell division, 1357; effect of, upon growth and swelling of plants, 1314.
Tetanus, effect of magnesium sulphate upon, 1307.
Tetany, guanidine, salt content of serum in, 1373.
Thymus, influence upon organism, 1308.
Thyroidectomy in Amphibia, 1349.
Tissue culture, method of measurement of growth in, 1298.

Toxin-antitoxin injections, immunity following, 1346.
Trinitrotoluene, poisoning from, 1366.
Tumor rate, effect of inbreeding on, 1330.
Typhoid, differentiation of, and paratyphoid *A* and *B*, 1304.
Tyramin, circulatory effects of, 1292.

Urea content of blood, 1343; splitting ferments, absence of, in tissues, 1293.
Urethra, absorptive power of, for drugs, 1312.
Uridin phosphoric acid, 1299.

Viability, effect upon, by transfer from stronger to weaker solutions, 1365.
Vegetables, dried, nitrogen and calcium content of, 1347.

PROCEEDINGS

OF THE

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

EIGHTY-FIFTH MEETING

CORNELL UNIVERSITY

MEDICAL COLLEGE

NEW YORK CITY

OCTOBER 17, 1917

AND

EIGHTEENTH MEETING

PACIFIC COAST BRANCH

SAN FRANCISCO, CALIFORNIA

OCTOBER 3, 1917

VOLUME XV

No. 1

NEW YORK

1917

CONTENTS.

LUDWIG KAST, V. C. MYERS AND EMMA L. WARDELL: The estimation of cholesterol in blood. 105 (1283).

JOHN AUER AND ISRAEL S. KLEINER: Morphin hyperglycaemia as a test for pancreatic deficiency. 106 (1284).

T. H. MORGAN: Demonstration of the effects of castration on Seabright Cockerels. 107 (1285).

I. S. KLEINER AND S. J. MELTZER: The effect of painting the pancreas with adrenalin on glycaemia and glycosuria. 108 (1286).

WILLIAM J. GIES AND COLLABORATORS: Studies of dental caries, with special reference to internal secretions in their relation to the development and condition of dental enamel. 109 (1287).

WILLIAM SALANT AND A. M. SWANSON: Diet and renal activity in tartrate nephritis. 110 (1288).

WILLIAM SALANT AND HELENE CONNET: Experiments with an isomer of caffein. 111 (1289).

OLAF BERGEIM: The determination and significance of intragastric conductance. 112 (1290).

JOHN O. HALVERSON, HENRY K. MOHLER AND OLAF BERGEIM: The calcium content of the blood serum in certain pathological conditions. 113 (1291).

A. W. HEWLETT: Circulatory effects of tyramin. 114 (1292).

AHMED E. SHEVKY (by invitation): Absence of urea-splitting ferments in the animal tissues. 115 (1293).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

PRESIDENT—William J. Gies, College of Physicians and Surgeons.

VICE-PRESIDENT—John Auer, Rockefeller Institute for Medical Research.

SECRETARY-TREASURER—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—E. F. Dubois, Cornell University Medical College, and George B. Wallace, University and Bellevue Hospital Medical College.

MANAGING EDITOR—The Secretary-Treasurer, 338 East 26th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

EIGHTY-SIXTH MEETING

NEW YORK POST GRADUATE MEDICAL SCHOOL

NEW YORK CITY

NOVEMBER 21, 1917

VOLUME XV

No. 2

NEW YORK

1917

CONTENTS

N. W. Janney and V. I. Isaacson: A blood sugar tolerance test. 116 (1294).

Anton R. Rose (by invitation): The inversion and determination of cane sugar. 117 (1295).

John A. Killian (by invitation): The influence of operative anesthesia and alkali therapy on the diastatic activity and sugar of human blood. 118 (1296).

A. R. Moore: Acid development as the result of injury in nervous tissue. 119 (1297).

H. Krigel (by invitation): A new method of tissue culture for accurate and rapid measurements of the growth. 120 (1298).

P. A. Levene: Uridin and cytidin-phosphoric acid. 121 (1299).

Isaac Levin and M. Levine: The influence of X-Rays on the development of the crown gall. 122 (1300).

K. George Falk and Kanematsu Sugiura: The two forms of glycine. 123 (1301).

David I. Macht (by invitation): On the absorption of apomorphin and morphin through unusual channels. 124 (1302).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

President—William J. Gies, College of Physicians and Surgeons.

Vice-President—John Auer, Rockefeller Institute for Medical Research.

Secretary-Treasurer—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—E. F. Dubois, Cornell University Medical College, and George B. Wallace, University and Bellevue Hospital Medical College.

Managing Editor—The Secretary-Treasurer, 338 East 26th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

EIGHTY-SEVENTH MEETING

ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH

NEW YORK CITY

DECEMBER 19, 1917

VOLUME XV

No. 3

NEW YORK

1917

CONTENTS

HERBERT D. TAYLOR AND J. HAROLD AUSTIN: The solvent action of antiseptics on necrotic tissue. 125 (1303).

FLORENCE HULTON-FRANKEL AND KATHERINE MACDONALD (by invitation): Differentiation of typhoid, paratyphoid A and B by means of a dextrin-inosite medium. 126 (1304).

P. A. LEVENE AND C. J. WEST: Hydrolecithin and its bearing on the constitution of cephalin. 127 (1305).

C. J. WIGGERS: Fat emboli and shock. 128 (1306).

J. AUER AND S. J. MELTZER: Lantern slide demonstration of the effect of magnesium sulphate upon tetanus. 129 (1307).

E. UHLENHUTH: Does the thymus glan dof mammals when given as food to Amphibians exert any specific influence upon the organism? 130 (1308).

A. I. RINGER: Demonstration of blood from an extreme case of lipemia in diabetes mellitus. 131 (1309).

GLENN E. CULLEN AND JAMES H. AUSTIN: A note on the preparation of Dakin's hypochlorite solution. 132 (1310).

HAROLD L. AMOSS AND FREDERICK EBERSON: Therapeutic experiments with Rosenow's antipoliomyelitic serum. 133 (1311).

DAVID I. MACHT: On the comparative absorptive power for drugs of the bladder and urethra (male). 134 (1312).

DAVID I. MACHT, S. ISAACS AND J. P. GREENBERG: On the influence of some opiates and antipyretics on the field of vision. 135 (1313).

D. T. MACDOUGAL: The relation of growth and swelling of plants and biocolloids to temperature. 136 (1314).

JACQUES LOEB: The chemical basis of morphological polarity in regeneration. 137 (1315).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

PRESIDENT—William J. Gies, College of Physicians and Surgeons.

VICE-PRESIDENT—John Auer, Rockefeller Institute for Medical Research.

SECRETARY-TREASURER—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—E. F. Dubois, Cornell University Medical College, and George B. Wallace, University and Bellevue Hospital Medical College.

MANAGING EDITOR—The Secretary-Treasurer, 338 East 26th St., New York City.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, D. J. Edwards, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H-Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Wil. liams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, W. Coleman, E. F. DuBois, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, Richard Weil, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, R. L. Kahn, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, J. W. Draper, Edward K. Dunham, E. M. Ewing, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, C. J. West, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 E. Broadway, N. Y. City.—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (New Haven).—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs

Carnegie Institution of Washington. (Station for Experimental Evolution, Cold Spring Harbor, N. Y.).—A. M. Banta, Charles B. Davenport, O. Riddle. *(Desert Laboratory, Tucson, Ariz.).*—D. T. MacDougal. *(Tortugas, Florida).*—Alfred G. Mayer.

State Boards of Health. Georgia (Atlanta).—Katharine R. Collins. *New York (Albany).*—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (Cincinnati, Ohio).—Martin H. Fischer. *Government Hospital for Insane (Washington, D. C.).*—J. A. F. Pfeiffer. *Mercy (Pittsfield, Mass.).*—Thomas Flournoy. *Peter Bent Brigham (Boston).*—Francis W. Peabody. *Presbyterian (Phila.).*—Ralph Pemberton. *Robert Brigham (Boston).*—F. M. McCrudden. *Royal Victoria (Montreal).*—Horst Oertel. *Western Pennsylvania (Pittsburgh).*—Max Kahn, Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Morris (Chicago).*—Max Morse. *Pasteur (Paris).*—Edna Steinhardt Harde. *Phipps (Philadelphia)*—Paul A. Lewis. *Rockefeller (Princeton.)*—Rhoda Erdmann, F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—T. C. Burnett, J. V. Cooke, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V Lamar. *Harvard.*—Herman M. Adler, Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, T. C. Janeway, H. S. Jennings, Walter Jones, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Leland Stanford.*—Thomas Addis, A. C. Crawford, E. C. Dickson, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.

Cambridge. Mass., 60 Buckingham Street.—W. J. Osterhout.

Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.

Kastanienbaum, Switzerland.—Fritz Schwyzer.

New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.

Otisville, N. Y.—B. White.

Rosebank, N. Y.—Oscar Teague.

Upper Montclair, N. J.—George Peirce.

Winnipeg, Canada.—F. J. Birchard.

Members present at the eighty-seventh meeting:

Amoss, Auer, Austin, Cole, Edwards, Falk, Flexner, Gies, Githens, Goldfarb, Jackson, Kleiner, Lambert, Levene, Loeb, Lusk, Meyer, A. L., Meyer, G. M., Pike, Ringer, Terry, Uhlenhuth, Van Slyke, West, Wiggers.

Dates of the next two meetings:

January 16, 1918–February 20, 1918.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, D. J. Edwards, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, W. Coleman, E. F. DuBois, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, Richard Weil, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, R. L. Kahn, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz. M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, J. W. Draper, Edward K. Dunham, E. M. Ewing, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, C. J. West, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 *E. Broadway, N. Y. City.*—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (New Haven).—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs.

Carnegie Institution of Washington. (Station for Experimental Evolution, Cold Spring Harbor, N. Y.).—A. M. Banta, Charles B. Davenport, O. Riddle. *(Desert Laboratory, Tucson, Ariz.).*—D. T. MacDougal. *(Tortugas, Florida).*—Alfred G. Mayer.

State Boards of Health. Georgia (Atlanta).—Katharine R. Collins. *New York (Albany).*—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (Cincinnati, Ohio).—Martin H. Fischer. *Government Hospital for Insane (Washington, D. C.).*—J. A. F. Pfeiffer. *Mercy (Pittsfield, Mass.).*—Thomas Flournoy. *Peter Bent Brigham (Boston).*—Francis W. Peabody. *Presbyterian (Phila.).*—Ralph Pemberton. *Robert Brigham (Boston).*—F. M. McCrudden *Royal Victoria (Montreal).*—Horst Oertel. *Western Pennsylvania (Pittsburgh).*—Max Kahn, Jacob Rosenbloom.

Institutes. Gratwick Laboratory (*Buffalo*).—G. H. A. Clowes, H. R. Gaylord. *Morris* (*Chicago*).—Max Morse. *Pasteur* (*Paris*).—Edna Steinhardt Harde. *Phipps* (*Philadelphia*)—Paul A. Lewis. *Rockefeller* (*Princeton.*)—Rhoda Erdmann, F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar* (*Philadelphia*).—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (*Washington, D. C.*).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Medical Corps* (*Fort Sam Houston, Texas*).—J. F. Siler. *Ohio River Investigation* (*Ohio*).—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—T. C. Burnett, J. V. Cooke, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V Lamar. *Harvard.*—Herman M. Adler, Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann, *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, T. C. Janeway, H. S. Jennings, Walter Jones, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Leland Stanford.*—Thomas Addis, A. C. Crawford, E. C. Dickson, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W, Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill* (*Montreal*).—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California* (*Los Angeles*).—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt* (*Nashville*).—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington* (*St. Louis*).—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve* (*Cleveland*).—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.

Cambridge, Mass., 60 Buckingham Street.—W. J. Osterhout.

Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.

Kastanienbaum, Switzerland.—Fritz Schwyzer.

New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.

Otisville, N. Y.—B. White.

Rosebank, N. Y.—Oscar Teague.

Upper Montclair, N. J.—George Peirce.

Winnipeg, Canada.—F. J. Birchard.

Members present at the eighty-sixth meeting:

Auer, Falk, Funk, Gies, Githens, Goldfarb, Jackson, Janney, Kleiner, Lambert, Levine, Meyer, G. M., Moore, Myers, V. C.

Members elected at the eighty-sixth meeting:

David I. Macht, George B. Roth.

Dates of the next two meetings:

December 19, 1917–January 16, 1918.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, D. J. Edwards, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, W. Coleman, E. F. DuBois, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, Richard Weil, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, R. L. Kahn, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, J. W. Draper, Edward K. Dunham, E. M. Ewing, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, C. J. West, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 *E. Broadway, N. Y. City.*—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (*New Haven*).—Thomas B. Osborne. *Maine* (*Orono*).—Raymond Pearl. *Maryland* (*Beltsville*).—E. B. Meigs

Carnegie Institution of Washington. (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*).—A. M. Banta, Charles B. Davenport, O. Riddle. (*Desert Laboratory, Tucson, Ariz.*).—D. T. MacDougal. (*Tortugas, Florida*).—Alfred G. Mayer.

State Boards of Health. Georgia (*Atlanta*).—Katharine R. Collins. *New York* (*Albany*).—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (*Cincinnati, Ohio*).—Martin H. Fischer. *Government Hospital for Insane* (*Washington, D. C.*).—J. A. F. Pfeiffer. *Mercy* (*Pittsfield, Mass.*).—Thomas Flournoy. *Peter Bent Brigham* (*Boston*).—Francis W. Peabody. *Presbyterian* (*Phila.*).—Ralph Pemberton. *Robert Brigham* (*Boston*).—F. M. McCrudden. *Royal Victoria* (*Montreal*).—Horst Oertel. *Western Pennsylvania* (*Pittsburgh*).—Max Kahn, Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Morris (Chicago).*—Max Morse. *Pasteur (Paris).*—Edna Steinhardt Harde. *Phipps (Philadelphia)*—Paul A. Lewis. *Rockefeller (Princeton.)*—Rhoda Erdmann, F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—T. C. Burnett, J. V. Cooke, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V. Lamar. *Harvard.*—Herman M. Adler, Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann, *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, T. C. Janeway, H. S. Jennings, Walter Jones, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Leland Stanford.*—Thomas Addis, A. C. Crawford, E. C. Dickson, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.

Cambridge, Mass., 60 Buckingham Street.—W. J. Osterhout.

Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.

Kastanienbaum, Switzerland.—Fritz Schwyzer.

New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.

Otisville, N. Y.—B. White.

Rosebank, N. Y.—Oscar Teague.

Upper Montclair, N. J.—George Peirce.

Winnipeg, Canada.—F. J. Birchard.

Members present at the eighty-fifth meeting:

Auer, Benedict, Gies, Githens, Jackson, Kleiner, Lusk, Meltzer, Morgan, Myers, V. C., Ringer, Wiggers.

Members present at the eighteenth meeting of the Pacific Coast Branch:

Addis, Burnett, Cooke, Dickson, Hewlett, Hooper, Hurwitz, Walker, Whipple.

Members elected at the eighty-fifth meeting:

Walter C. Alvarez, G. D. Barnett, George W. Corner, Harold K. Faber, F. L. Gates, Maurice Hope Givens, C. Lundsgaard.

Dates of the next two meetings:

November 21, 1917–December 19, 1917.

PROCEEDINGS

OF THE

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

EIGHTY-EIGHTH MEETING

COLLEGE OF PHYSICIANS AND SURGEONS

JANUARY 16, 1918

NEW YORK CITY

VOLUME XV

No. 4

NEW YORK

1918

CONTENTS

A. B. Stout (*by invitation*): Experimental studies of self-incompatibilities in fertilization. 138 (1316).

I. Strauss and I. Friesner (*by invitation*): A demonstration of cerebellar and cerebral lesions in dogs. 139 (1317).

H. C. Sherman and D. E. Neun: Amylase and protease action of some pancreas preparations. 140 (1318).

Helen C. Coombs and F. H. Pike: The rôle of afferent impulses in the control of respiratory movements. 141 (1319).

Oscar Riddle: A case of hereditary ataxia (?) in pigeons. 142 (1320).

Martha Wollstein and S. J. Meltzer: Chemical pneumonia. 143 (1321).

Eugene L. Porter (*by invitation*): Changes in reflex thresholds following experimental shock from intestinal manipulation. 144 (1322).

David I. Macht, S. Isaacs and J. P. Greenberg: On the influence of some antipyretics on the neuro-muscular coordination test of "tapping." 145 (1323).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

President—William J. Gies, College of Physicians and Surgeons.

Vice-President—John Auer, Rockefeller Institute for Medical Research.

Secretary-Treasurer—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—E. F. Dubois, Cornell University Medical College, and George B. Wallace, University and Bellevue Hospital Medical College.

Managing Editor—The Secretary-Treasurer, 338 East 26th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

EIGHTY-NINTH MEETING

COLLEGE OF THE CITY OF NEW YORK

NEW YORK CITY

FEBRUARY 20, 1918

AND

NINETEENTH MEETING

PACIFIC COAST BRANCH

SAN FRANCISCO, CALIFORNIA

FEBRUARY 6, 1918

VOLUME XV

No. 5

NEW YORK

1918

CONTENTS

DAVID I. MACHT: On the relation of the chemical structure of the opium alkaloids to their effect on smooth muscle, and the discovery of a new therapeutic agent as a consequence thereof. 146 (1324).

MAURICE H. GIVENS: Studies in calcium and magnesium metabolism. Further observations on the effect of acid and dietary factors. 147 (1325).

C.-E. A. WINSLOW AND I. S. FALK: Studies on salt action. I. Effect of calcium and sodium salts upon the viability of the colon bacillus in water. 148 (1326).

N. W. JANNEY AND V. I. ISAACSON: Protein sparing by glucose in experimental diabetes. 149 (1327).

T. S. GITHENS AND S. J. MELTZER: Studies in experimental shock. 150 (1328).

LAFAYETTE B. MENDEL AND THOMAS B. OSBORNE: Further observations on the nutritive factors in animal tissues. 151 (1329).

LEO LOEB AND A. E. C. LATHROP: The effect of continued inbreeding on the tumor rate in mice. 152 (1330).

MAX KAHN: Sulpho-conjugation as a test of hepatic function. 153 (1331).

J. C. TORREY AND ALFRED F. HESS: The relation of the intestinal flora to scurvy of guinea-pigs and of infants. 154 (1332).

T. ADDIS AND A. E. SHEVKY: Sources of error in the estimation of dextrose by the calorimetric picrate method. 155 (1333).

THEODORE C. BURNETT: Does the liver secrete a catalase accelerator? 156 (1334).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

PRESIDENT—William J. Gies, College of Physicians and Surgeons.

VICE-PRESIDENT—John Auer, Rockefeller Institute for Medical Research.

SECRETARY-TREASURER—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—George B. Wallace, University and Bellevue Hospital Medical College, and Henry C. Sherman, Columbia University.

MANAGING EDITOR—The Secretary-Treasurer, 338 East 26th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

NINETIETH MEETING

PRESBYTERIAN HOSPITAL

MARCH 20, 1918

NEW YORK CITY

VOLUME XV

No. 6

NEW YORK

1918

CONTENTS

ISRAEL S. KLEINER: The rate of dialysis of diabetic blood-sugar. 157 (1335).

ALFRED F. HESS AND LESTER J. UNGER: Experiments on the scurvy of guinea pigs. 158 (1336).

MIRIAM OLMSTEAD: Types of pneumococci found in the mouths of surgical cases before operation. 159 (1337).

DANA W. ATCHLEY (*by invitation*): Renal action in acute nephritis. 160 (1338).

ALBERT A. EPSTEIN: The application of the Auer-Kleiner morphine test in human diabetes. 161 (1339).

THEODORE KUTTNER (*by invitation*): A method for determining minute quantities of nitrogen in nitrogenous substances. 162 (1340).

WARREN COLEMAN: Asynchronism of the respiratory movements in lobar pneumonia. 163 (1341).

CHARLES WEISS AND JOHN A. KOLMER: A skin reaction to pneumotoxin. 164 (1342).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

PRESIDENT—William J. Gies, College of Physicians and Surgeons.

VICE-PRESIDENT—John Auer, Rockefeller Institute for Medical Research.

SECRETARY-TREASURER—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—George B. Wallace, University and Bellevue Hospital Medical College, and Henry C. Sherman, Columbia University.

MANAGING EDITOR—The Secretary-Treasurer, 338 East 26th St., New York City.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H-Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Wil. iams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, E. F. DuBois, D. J. Edwards, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *Beth Israel.*—Max Kahn. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 *E. Broadway, N. Y. City.*—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (New Haven).—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs

Carnegie Institution of Washington. (Station for Experimental Evolution, Cold Spring Harbor, N. Y.).—A. M. Banta, Charles B. Davenport, O. Riddle. *(Desert Laboratory, Tucson, Ariz.).*—D. T. MacDougal. *(Tortugas, Florida).*—Alfred G. Mayer.

State Boards of Health. Georgia (Atlanta).—Katharine R. Collins. *New York (Albany).*—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (Cincinnati, Ohio).—Martin H. Fischer. *Government Hospital for Insane (Washington, D. C.).*—J. A. F. Pfeiffer. *Mercy (Pittsfield, Mass.).*—Thomas Flournoy. *Peter Bent Brigham (Boston).*—Francis W. Peabody. *Presbyterian (Phila.).*—Ralph Pemberton. *Robert Brigham (Boston).*—F. M. McCrudden. *Royal Victoria (Montreal).*—Horst Oertel. *Western Pennsylvania (Pittsburgh).*—Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Juvenile Psychopathic (Chicago).*—Herman M. Adler. *Morris (Chicago).*—Withrow Morse. *Phipps (Philadelphia).*—Paul A. Lewis. *Rockefeller (Princeton).*—F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Department Laboratory (Atlanta, Ga.).*—R. L. Kahn. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—Walter C. Alvarez, T. C. Burnett, J. V. Cooke, George W. Corner, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson, *Georgia.*—Richard V. Lamar. *Harvard.*—Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, Dr. W. J. V. Osterhout, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, H. S. Jennings, Walter Jones, David I. Macht, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Kansas.*—George B. Roth. *Leland Stanford.*—Thomas Addis, George D. Barnett, A. C. Crawford, E. C. Dickson, Harold K. Faber, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Rhoda Erdmann, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.
Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.
Kastanienbaum, Switzerland.—Fritz Schwyzer.
New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.
Otisville, N. Y.—B. White.
Rosebank, N. Y.—Oscar Teague.
Silver City, New Mexico.—E. M. Ewing,
Upper Montclair, N. J.—George Peirce.
Washington, D. C., 3315 Wisconsin Ave.—C. J. West.
Winnipeg, Canada.—F. J. Birchard.

Members present at the ninetieth meeting:

Coleman, Epstein, Gies, Githens, Goldfarb, Hess, Jackson, Kleiner, Lambert, Lieb, Loeb, Meltzer, Northrop, Olmstead, Salant, Sherman, Uhlenhuth.

Members elected at the ninetieth meeting:

Arthur Knudson, D. E. Jackson, C. P. Sherwin, Arthur W. Thomas.

Dates of the next two meetings:

April 17, 1918–May 15, 1918

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, E. F. DuBois, D. J. Edwards, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, Richard Weil, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *Beth Israel.*—Max Kahn. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 E. Broadway, N. Y. City.—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (New Haven).—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs.

Carnegie Institution of Washington. (Station for Experimental Evolution, Cold Spring Harbor, N. Y.).—A. M. Banta, Charles B. Davenport, O. Riddle. *(Desert Laboratory, Tucson, Ariz.).*—D. T. MacDougal. *(Tortugas, Florida).*—Alfred G. Mayer.

State Boards of Health. Georgia (Atlanta).—Katharine R. Collins. *New York (Albany).*—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (Cincinnati, Ohio).—Martin H. Fischer. *Government Hospital for Insane (Washington, D. C.).*—J. A. F. Pfeiffer. *Mercy (Pittsfield, Mass.).*—Thomas Flournoy. *Peter Bent Brigham (Boston).*—Francis W. Peabody. *Presbyterian (Phila.).*—Ralph Pemberton. *Robert Brigham (Boston).*—F. M. McCrudden. *Royal Victoria (Montreal).*—Horst Oertel. *Western Pennsylvania (Pittsburgh).*—Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Juvenile Psychopathic (Chicago).*—Herman M. Adler. *Morris (Chicago).*—Withrow Morse. *Pasteur (Paris).*—Edna Steinhardt Harde. *Phipps (Philadelphia).*—Paul A. Lewis. *Rockefeller (Princeton).*—Rhoda Erdmann, F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Department Laboratory (Atlanta, Ga.).*—R. L. Kahn. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—Walter C. Alvarez, T. C. Burnett, J. V. Cooke, George W. Corner, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V. Lamar. *Harvard.*—Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, Dr. W. J. V. Osterhout, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, H. S. Jennings, Walter Jones, David I. Macht, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Kansas.*—George B. Roth. *Leland Stanford.*—Thomas Addis, George D. Barnett, A. C. Crawford, E. C. Dickson, Harold K. Faber, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.
Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.
Kastanienbaum, Switzerland.—Fritz Schwyzer.
New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.
Otisville, N. Y.—B. White.
Rosebank, N. Y.—Oscar Teague.
Silver City, New Mexico.—E. M. Ewing,
Upper Montclair, N. J.—George Peirce.
Washington, D. C., 3315 Wisconsin Ave.—C. J. West.
Winnipeg, Canada.—F. J. Birchard.

Members present at the eighty-ninth meeting:

Auer, Benedict, Gies, Githens, Givens, Goldfarb, Hess, Jackson, Janney, Kober, Macht, Meltzer, Mendel, Meyer, A. L., Peirce, Sherman, Wadsworth, Wiggers, Winslow.

Members present at the nineteenth meeting of the Pacific Coast Branch:

Addis, Alvarez, Burnett, Dickson, Evans, Hooper, Whipple.

Member elected at the eighty-ninth meeting:

A. R. Rose.

Dates of the next two meetings:

March 20, 1918–April 17, 1918.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, D. J. Edwards, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, W. Coleman, E. F. DuBois, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, Richard Weil, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, R. L. Kahn, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz. M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, J. W. Draper, Edward K. Dunham, E. M. Ewing, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, C. J. West, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 *E. Broadway, N. Y. City.*—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (*New Haven*).—Thomas B. Osborne. *Maine* (*Orono*).—Raymond Pearl. *Maryland* (*Beltsville*).—E. B. Meigs.

Carnegie Institution of Washington. (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*).—A. M. Banta, Charles B. Davenport, O. Riddle. (*Desert Laboratory, Tucson, Ariz.*).—D. T. MacDougal. (*Tortugas, Florida*).—Alfred G. Mayer.

State Boards of Health. Georgia (*Atlanta*).—Katharine R. Collins. *New York* (*Albany*).—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (*Cincinnati, Ohio*).—Martin H. Fischer. *Government Hospital for Insane* (*Washington, D. C.*).—J. A. F. Pfeiffer. *Mercy* (*Pittsfield, Mass.*).—Thomas Flournoy. *Peter Bent Brigham* (*Boston*).—Francis W. Peabody. *Presbyterian* (*Phila.*).—Ralph Pemberton. *Robert Brigham* (*Boston*).—F. M. McCrudden. *Royal Victoria* (*Montreal*).—Horst Oertel. *Western Pennsylvania* (*Pittsburgh*).—Max Kahn, Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Morris (Chicago).*—Max Morse. *Pasteur (Paris).*—Edna Steinhardt Harde. *Phipps (Philadelphia)*—Paul A. Lewis. *Rockefeller (Princeton.)*—Rhoda Erdmann, F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—T. C. Burnett, J. V. Cooke, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V Lamar. *Harvard.*—Herman M. Adler, Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann, *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, T. C. Janeway, H. S. Jennings, Walter Jones, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Leland Stanford.*—Thomas Addis, A. C. Crawford, E. C. Dickson, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W, Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.

Cambridge. Mass., 60 Buckingham Street.—W. J. Osterhout.

Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.

Kastanienbaum, Switzerland.—Fritz Schwyzer.

New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.

Otisville, N. Y.—B. White.

Rosebank, N. Y.—Oscar Teague.

Upper Montclair, N. J.—George Peirce.

Winnipeg, Canada.—F. J. Birchard.

Members present at the eighty-eighth meeting:

Auer, Edwards, Falk, Coleman, Gies, Jackson, Kleiner, Lambert, Lieb, Meltzer, Meyer, A. L., Myers, Pike, Uhlenhuth, Sherman, Riddle, Wadsworth, Wiggers, Wollstein.

Dates of the next two meetings:

February 20, 1918–March 20, 1918

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

NINETY-FIRST MEETING

UNIVERSITY AND BELLEVUE HOSPITAL

MEDICAL COLLEGE

NEW YORK CITY

APRIL 17, 1918

AND

TWENTIETH MEETING

PACIFIC COAST BRANCH

SAN FRANCISCO, CALIFORNIA

APRIL 8, 1918

VOLUME XV

No. 7

NEW YORK

1918

CONTENTS.

LUDWIG KAST AND EMMA M. WARDELL: The urea content of the blood. 165 (1343).

RHODA ERDMANN: Production of transplantable growth. 166 (1344).

H. L. ABRAMSON (*by invitation*): Immunization of monkeys against poliomyelitis. 167 (1345).

WILLIAM H. PARK: Persistence of immunity following toxin-antitoxin injections. 168 (1346).

MAURICE H. GIVENS: The composition of dried vegetables with special reference to their nitrogen and calcium content. 169 (1347).

BARNETT COHEN (*by invitation*): Observations on the production of experimental scurvy in guinea pig. 170 (1348).

E. R. HOSKINS AND M. M. HOSKINS: Further experiments with thyroidectomy in Amphibia. 171 (1349).

JOHN AUER: Generalized analgesia in cats after exposure to a war-gas, $(CH_3)_2SO_4$. 172 (1350).

JOHN AUER: Localized pulmonary edema in cats after inhalation of a war-gas, $(CH_3)_2SO_4$. 173 (1351).

G. H. A. CLOWES: On the electrical resistance and permeability of tumor tissues. 174 (1352).

G. H. A. CLOWES: On the action exerted by antagonistic electrolytes on the electrical resistance and permeability of emulsion membranes. 175 (1353).

ROBERT A. LAMBERT AND S. S. SAMUELS: The relationship of the leucocyte count and bone-marrow changes in acute lobar pneumonia. 176 (1354)

W. OPHÜLS AND ELMER W. SMITH: Anatomical changes produced by repeated intravenous injections of streptococci in rabbits. 177 (1355).

LILLIAN J. ELLEFSON AND CARL L. A. SCHMIDT: On serum proteins. 178 (1356).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

PRESIDENT—William J. Gies, College of Physicians and Surgeons.

VICE-PRESIDENT—John Auer, Rockefeller Institute for Medical Research.

SECRETARY-TREASURER—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—George B. Wallace, University and Bellevue Hospital Medical College, and Henry C. Sherman, Columbia University.

MANAGING EDITOR—The Secretary-Treasurer, 338 East 26th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

NINETY-SECOND MEETING

COLUMBIA UNIVERSITY

SCHERMERHORN HALL

MAY 15, 1918

NEW YORK CITY

VOLUME XV

No. 8

NEW YORK

1918

CONTENTS.

H. H. Laughlin (By invitation): The dynamics of cell-division. 179 (1357).

Barnett Cohen and Lafayette B. Mendel: Diet and roughage in relation to the experimental scurvy of guinea pigs. 180 (1358).

C.-E. A. Winslow and David Greenberg: The effect of the respiration of putrid gases upon the growth of guinea pigs. 181 (1359).

A. K. Balls and José D. Moral: Studies on the action of certain antiseptics, toxic salts and alkaloids against the bacteria and protozoa of the intestine of the rabbit. 182 (1360).

Maurice H. Givens and Barnett Cohen: The influence of cooking and drying cabbage on its antiscorbutic properties for guinea pigs. 183 (1361).

T. S. Githens and S. J. Meltzer: The prevention of blood clotting by Dakin's sodium hypochlorite solution. 184 (1362).

A. L. Meyer and S. J. Meltzer: The minimum number of respirations sufficient to maintain life. 185 (1363).

Jacques Loeb: The action of neutral salts on the osmotic pressure and other qualities of gelatin. 186 (1364).

C.-E. A. Winslow and I. S. Falk: Studies on salt action. II. The effect of transfer from stronger to weaker salt solutions upon the viability of bacteria in water. 187 (1365).

Rudolph Kramer and Harold Meierhof. (*By Invitation*): Experimental trinitro-toluene poisoning. 188 (1366).

M. Dresbach and W. L. Chandler: The toxic action of nitrobenzene with special reference to the cerebellum. 189 (1367).

Max Kahn: A method for the simultaneous fractional analyses of gastric and duodenal contents. 190 (1368).

Montrose T. Burrows and Clarence A. Neymann: Studies on the metabolism of cells in vitro. The toxicity of dipeptids for embryonic chicken cells. 191 (1369).

L. S. N. Walsh and Leo Loeb: A note on the healing in and migration of foreign bodies in the animal body. 192 (1370).

Alfred F. Hess and Lester J. Unger: Experiments on antiscorbutics. Report of an antiscorbutic for intravenous use. 193 (1371).

C. K. Watanabe and V. C. Myers: A delicate method of determining invert activity. 194 (1372).

C. K. Watanabe: The phosphate and calcium content of serum in the condition of guanidine tetany. 195 (1373).

L. Baumann and Thorsten Ingvaldsen: An oxidation product of creatine. 196 (1374).

L. Baumann and Thorsten Ingvaldsen: Concerning carnosine and its synthesis. 197 (1375).

The Proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesday of the months of October to May inclusive. A volume of the Proceedings consists of the numbers issued during an academic year.

The price of Vols. I, II and III is four dollars each, of Vol. IV to current volume is two dollars each, postage prepaid. The price of copies of the proceedings of any meeting is thirty cents each, postage prepaid. Subscriptions are payable in advance.

President—William J. Gies, College of Physicians and Surgeons.

Vice-President—John Auer, Rockefeller Institute for Medical Research.

Secretary-Treasurer—Holmes C. Jackson, University and Bellevue Hospital Medical College.

Additional members of the Council—George B. Wallace, University and Bellevue Hospital Medical College, and Henry C. Sherman, Columbia University.

Managing Editor—The Secretary-Treasurer, 338 East 26th St., New York City.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—A. K. Balls, Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, George Draper, Haven Emerson, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, Arthur W. Thomas, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, E. F. DuBois, D. J. Edwards, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, Charles R. Stockard, W. C. Thro, John C. Torrey, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee, Carl P. Sherwin.

Hospitals, Bellevue.—Charles Norris. *Beth Israel.*—Max Kahn. *Memorial Hospital.*—H. H. Janeway. *Montefiore Home.*—George Fahr, Nelson W. Janney, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead, Walter W. Palmer. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Post-Graduate Medical School.—Ludwig Kast, John A. Killian, W. J. MacNeal, V. C. Myers, Anton R. Rose, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, F. L. Gates, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 *E. Broadway, N. Y. City.*—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (*New Haven*).—Thomas B. Osborne. *Maine* (*Orono*).—Raymond Pearl. *Maryland* (*Beltsville*).—E. B. Meigs.

Carnegie Institution of Washington. (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*).—A. M. Banta, Charles B. Davenport, O. Riddle. (*Desert Laboratory, Tucson, Ariz.*).—D. T. MacDougal. (*Tortugas, Florida*).—Alfred G. Mayer.

State Boards of Health. New York (*Albany*).—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (*Cincinnati, Ohio*).—Martin H. Fischer. *Government Hospital for Insane* (*Washington, D. C.*).—J. A. F. Pfeiffer. *Mercy* (*Pittsfield, Mass.*).—Thomas Flournoy. *Peter Bent Brigham* (*Boston*).—Francis W. Peabody. *Presbyterian* (*Phila.*).—Ralph Pemberton. *Robert Brigham* (*Boston*).—F. M. McCrudden. *Royal Victoria* (*Montreal*).—Horst Oertel. *Western Pennsylvania* (*Pittsburgh*).—Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Juvenile Psychopathic (Chicago).*—Herman M. Adler. *Morris (Chicago).*—Withrow Morse. *Phipps (Philadelphia).*—Paul A. Lewis. *Rockefeller (Princeton).*—F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Department Laboratory (Atlanta, Ga.).*—R. L. Kahn. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—Walter C. Alvarez, T. C. Burnett, J. V. Cooke, George W. Corner, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, C. K. Watanabe, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Copenhagen.*—Christen Lundsgaard. *Cornell.*—Melvin Dresbach, Sutherland Simpson. *Georgia.*—Richard V. Lamar. *Harvard.*—Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, W. J. V. Osterhout, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, H. S. Jennings, Walter Jones, David I. Macht, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Kansas.*—Bennet M. Allen, George B. Roth. *Leland Stanford.*—Thomas Addis, George D. Barnett, A. C. Crawford, E. C. Dickson, Harold K. Faber, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rochester.*—Maurice H. Givens, John R. Murlin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum, T. Brailsford Robertson. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Union University (Albany Medical College).*—Arthur Knudson. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen, B. T. Terry. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. T. Burrows, Joseph Erlanger, D. E. Jackson, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Rhoda Erdmann, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C.-E. A. Winslow, Lorande Loss Woodruff.

Calco Chemical Co., Bound Brook, N. J.—M. S. Fine.
Cambridge, England.—C. G. L. Wolf.
Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.
Cleveland, Ohio, 8803 Euclid Ave.—Katharine R. Collins.
Kastanienbaum, Switzerland.—Fritz Schwyzer.
New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.
Otisville, N. Y.—B. White.
Rosebank, N. Y.—Oscar Teague.
Silver City, New Mexico.—E. M. Ewing.
Upper Montclair, N. J.—George Peirce.
Washington, D. C., 3315 Wisconsin Ave.—C. J. West.
Winnipeg, Canada.—F. J. Birchard.
Germany.—Beutner, Reinhard.

Members present at the ninety-second meeting:

Gies, Githens, Goldfarb, Hess, Jackson, Lambert, Loeb, J., Meltzer, Meyer, A. L., Morgan, Myers, V. C., Peirce, Sherman, Stockard, Uhlenhuth, Wilson, Winslow.

Members elected at the ninety-second meeting:

J. A. Killian, B. M. Allen.

Dates of the next two meetings:

October 16, 1918–November 20, 1918.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

College of the City of New York.—W. W. Browne, A. J. Goldfarb, G. G. Scott, Thomas A. Storey.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, R. L. Cecil, Henry E. Crampton, George Draper, William J. Gies, J. Gardner Hopkins, R. A. Lambert, Frederic S. Lee, Isaac Levin, C. C. Lieb, W. F. Longcope, W. G. MacCallum, Thomas H. Morgan, B. S. Oppenheimer, Alwin M. Pappenheimer, F. H. Pike, E. L. Scott, Henry C. Sherman, M. J. Sittenfield, H. F. Swift, H. B. Williams, Edmund B. Wilson, Francis C. Wood, Hans Zinsser.

Cornell University Medical College.—Harold Bailey, Stanley R. Benedict, E. F. DuBois, D. J. Edwards, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey, C. J. Wiggers.

Fordham University School of Medicine.— A. O. Shaklee.

Hospitals, Bellevue.—Charles Norris. *Beth Israel.*—Max Kahn. *King's County.*—B. T. Terry. *Montefiore Home.*—George Fahr, Nelson W. Janney, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

New York City Departments. Education.—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, Haven Emerson, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—M. S. Fine, Ludwig Kast, W. J. MacNeal, V. C. Myers, R. M. Taylor.

New York University.—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson, H. H. Janeway, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

Rockefeller Institute for Medical Research.—Harold L. Amoss, John Auer, Reinhard Beutner, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

Brooklyn Botanic Garden.—C. Stuart Gager, O. E. White.

135 *E. 34th St., N. Y. City.*—E. E. Butterfield.

819 *Madison Avenue, N. Y. City.*—H. D. Dakin.

High School of Commerce Annex, 197 E. Broadway, N. Y. City.—Walter H. Eddy.

24 *East 48th St., N. Y. City.*—Cyrus W. Field.

215 *Manhattan Ave., N. Y. City.*—Casimir Funk.

56 *East 77th St., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

Non-Resident.

Agricultural Experiment Stations. Connecticut (New Haven).—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs.

Carnegie Institution of Washington. (Station for Experimental Evolution, Cold Spring Harbor, N. Y.).—A. M. Banta, Charles B. Davenport, O. Riddle. *(Desert Laboratory, Tucson, Ariz.).*—D. T. MacDougal. *(Tortugas, Florida).*—Alfred G. Mayer.

State Boards of Health. Georgia (Atlanta).—Katharine R. Collins. *New York (Albany).*—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

Hospitals. General (Cincinnati, Ohio).—Martin H. Fischer. *Government Hospital for Insane (Washington, D. C.).*—J. A. F. Pfeiffer. *Mercy (Pittsfield, Mass.).*—Thomas Flournoy. *Peter Bent Brigham (Boston).*—Francis W. Peabody. *Presbyterian (Phila.).*—Ralph Pemberton. *Robert Brigham (Boston).*—F. M. McCrudden. *Royal Victoria (Montreal).*—Horst Oertel. *Western Pennsylvania (Pittsburgh).*—Jacob Rosenbloom.

Institutes. Gratwick Laboratory (Buffalo).—G. H. A. Clowes, H. R. Gaylord. *Juvenile Psychopathic (Chicago).*—Herman M. Adler. *Morris (Chicago).*—Withrow Morse. *Phipps (Philadelphia).*—Paul A. Lewis. *Rockefeller (Princeton).*—F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

U. S. Departments. Agriculture (Washington, D. C.).—Carl L. Alsberg, William N. Berg, J. R. Mohler, William Salant. *Department Laboratory (Atlanta, Ga.).*—R. L. Kahn. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

Universities. Buffalo.—Herbert U. Williams. *California.*—Walter C. Alvarez, T. C. Burnett, J. V. Cooke, George W. Corner, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, T. Brailsford Robertson, Carl L. A. Schmidt, E. L. Walker, H. Wasteneys, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Clark.*—Ralph S. Lillie. *Cornell.*—Melvin Dresbach, Sutherland Simpson, *Georgia.*—Richard V. Lamar. *Harvard.*—Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, David L. Edsall, Otto Folin, Worth Hale, Reid Hunt, Dr. W. J. V. Osterhout, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, H. S. Jennings, Walter Jones, David I. Macht, Adolph Meyer, H. O. Mosenthal, E. A. Park, William H. Welch. *Kansas.*—George B. Roth. *Leland Stanford.*—Thomas Addis, George D. Barnett, A. C. Crawford, E. C. Dickson, Harold K. Faber, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, Otto C. Glaser, G. Carl Huber, Warren P. Lombard, Frederick G. Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. de B. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum. *Tufts.*—C. H. Bailey. *Tulane.*—Charles W. Duval. *Vanderbilt (Nashville).*—J. W. Jobling, W. F. Petersen. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. J. Burrows, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R. Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *West Virginia.*—W. H. Schultz. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Rhoda Erdmann, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C. K. Watanabe, C.-E. A. Winslow, Lorande Loss Woodruff.

Cambridge, England.—C. G. L. Wolf.
Chicago, Illinois, 104 S. Michigan Ave.—Solomon Strouse.
Kastanienbaum, Switzerland.—Fritz Schwyzer.
New Brunswick, N. J.—John F. Anderson, Isaac F. Harris.
Otisville, N. Y.—B. White.
Rosebank, N. Y.—Oscar Teague.
Silver City, New Mexico.—E. M. Ewing,
Upper Montclair, N. J.—George Peirce.
Washington, D. C., 3315 Wisconsin Ave.—C. J. West.
Winnipeg, Canada.—F. J. Birchard.

Members present at the ninety-first meeting:

Auer, Erdmann, Gies, Githens, Givens, Hess, Hoskins, Kast, Lambert, Myers, V. C., Jackson, Sherman, Uhlenhuth.

Members present at the twentieth meeting of the Pacific Coast Branch:

Alvarez, Burnett, Corner, Ophüls, Schmidt, Walker, Whipple.

Members elected at the ninety-first meeting:

A. K. Balls, W. W. Palmer.

Dates of the next two meetings:

May 15, 1918–October 16, 1918.

AMNH LIBRARY
100209755